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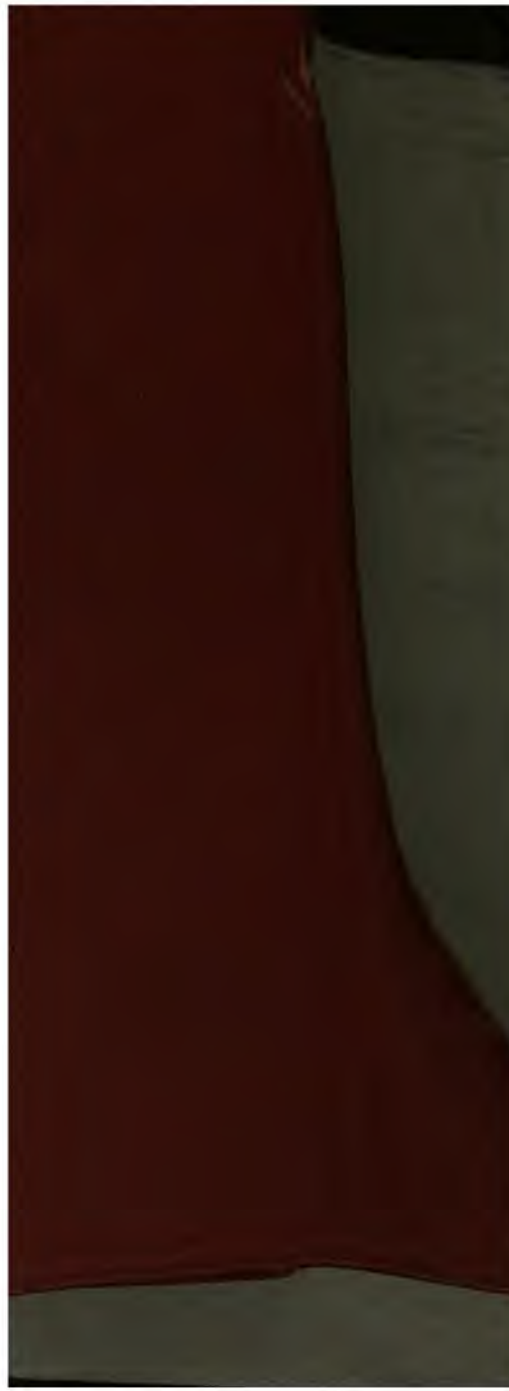

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TRANSACTIONS

OF

THE CLINICAL SOCIETY.

VOL. XXXVIII.

TRANSACTIONS

OF

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OF

LONDON.

VOLUME THE THIRTY-EIGHTH.

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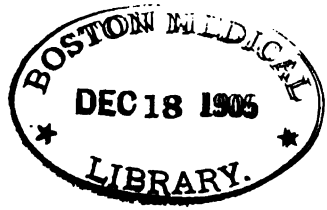

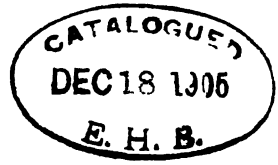

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THE present Volume comprises the Proceedings of the Society during its Thirty-eighth Session, October, 1904, to May, 1905.

The Council think it proper to state that the authors of the several communications are alone responsible for the statements, reasonings, and opinions contained in their respective papers.

20, HANOVER SQUARE, W.;
October 13th, 1905.

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1883 BALLANCE, CHARLES ALFRED, M.B., M.S., 106, Harley Street, W (C. 1894–6.) *Trans.* 3, *C.C.* 5.

1904 BALLANCE, H. A., 46, Prince of Wales Road, Norwich.

1888 BANHAM, HENRY FRENCH, M.D., Assington Vicarage, Colchester.

1868 BANTOCK, GEORGE GRANVILLE, M.D., 12, Granville Place, W.

1876 BARKER, ARTHUR E. J., 87, Harley Street, W. (C. 1883–5, V.P. 1901–3.) *Trans.* 18, *C.C.* 13.

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service, India.

1894 BARLING, HARRY GILBERT, 87, Cornwall Street, Newhall Street, Birmingham. (C. 1901–3.) *Trans.* 3.

1875 BARLOW, SIR THOMAS, Bart., K.C.V.O., M.D., 10, Wimpole Street, W. (C. 1880–2, 1891–4, S. 1888–90, V.P. 1895–8.) *Trans.* 11, *C.C.* 5.

1902 BARNARD, HAROLD L., 21, Wimpole Street, Cavendish Square, W. *Trans.* 2, *C.C.* 7.

1888 BARRS, A. G., M.D., 22, Park Place, Leeds.

1888 BARTON, EDWIN A., 56, Kensington Court, Kensington, W.

1891 BARTON, J. KINGSTON, 14, Ashburn Place, Courtfield Road, S.W. (C. 1901–4.)

1901 BARWELL, HAROLD SHUTTLEWORTH, 55, Wimpole Street, Cavendish Square, W.

O.M. BARWELL, RICHARD, 55, Wimpole Street, Cavendish Square, W. (C. 1872–5, V.P. 1877–9.) *Trans.* 21, *C.C.* 2.

O.M. **BASTIAN**, HENRY CHARLTON, M.D., F.R.S., 8A, Manchester Square, W. (C. 1876–8, V.P. 1891–3.) *Trans.* 7.

1896 BATTEN, FREDERICK E., M.D., 33, Harley Street, W. (C. 1903– .) *Trans.* 5, *C.C.* 8.

1899 BATTEN, RAYNER DERRY, M.D., B.S., 33, Harley Street, W. *C.C.* 1.

1888 BATTERHAM, JOHN WILLIAMS, M.B., B.S., Bank House, Grand Parade, St. Leonard's.

1886 BATTLE, WILLIAM HENRY, 49, Harley Street, W. (C. 1899–1902.) *Trans.* 7, *C.C.* 15.

1868 BÄUMLER, CHRISTIAN G. H., M.D., The University, Freiburg i/Br., Baden, Germany. *Trans.* 4.

1903 BEAUMONT, EDGAR, "Claremont," Gipsy Hill, Upper Norwood, S.E. *C.C.* 2.

1898 BEDDARD, ARTHUR PHILIP, M.D., 44, Seymour Street, Portman Square, W.

1880 **BEEVOR**, CHARLES EDWARD, M.D., 135, Harley Street, W. (C. 1890–1.) *Trans.* 2, *C.C.* 9.

1901 BEEVOR, SIR HUGH REEVE, Bart., M.D., 17, Wimpole Street, Cavendish Square, W. *C.C.* 3.

ELECTED

1884 BENHAM, F. LUCAS, M.D., Campden House, nr. Exeter, Semaphore, South Australia. *Trans.* 4.

1883 BENHAM, ROBERT FITZROY, 11, Earl's Court Square, S.W.

1874 BENNETT, Sir WILLIAM HENRY, K.C.V.O., 1, Chesterfield Street, W. (C. 1889, 1892–4, S. 1890–1, V.P. 1895–8.) *Trans.* 4, *C.C.* 3.

1896 BENTHAM, EDWARD COOPER, 10, Princes Street, Cavendish Square, W.

1889 BENTLEY, ARTHUR J. M., M.D., Mena House, Pyramids, Cairo, Egypt.

1882 BERRY, FREDERICK HAYCRAFT, M.D., Watford, Herts.

1885 BERRY, JAMES, B.S., 21, Wimpole Street, W. (C. 1898–1901.) *Trans.* 1.

1889 BIDWELL, LEONARD ARTHUR, 15, Upper Wimpole Street, W. (C. 1901–4.) *Trans.* 5, *C.C.* 1.

1882 BINDLEY, PHILIP HENRY, M.B. (Travelling).

1889 BISSHOPP, FRANCIS ROBERT BRYANT, M.A., M.D., B.S., Parham House, Tunbridge Wells.

1904 BOLTON, CHARLES, M.D., 16, Devonshire Street, Portland Place, W. *C.C.* 1.

1893 BOND, JAMES WILLIAM, M.D., 26, Harley Street, W. *C.C.* 2.

1898 BOSANQUET, WILLIAM CECIL, M.D, 117A, Harley Street, Cavendish Square, W.

1888 BOSTOCK, ROBERT ASHTON, Cefn Mor, Penmaen, Glamorganshire.

1900 BOUSFIELD, ARTHUR, M.B., The Cheal, Hornsey Lane, N.

1899 BOWLAN, MARCUS MARWOOD, M.B., B.S., The Infirmary, Raine Street, St. George-in-the-East, E.

1883 BOWLBY, ANTHONY A., C.M.G., 24, Manchester Square, W. (C. 1891–3, 1904– , S. 1901–4.) *Trans.* 6, *C.C.* 4.

1883 **Bowles**, ROBERT LEAMON, M.D., Prior's Mesne, Lydney, Gloucestershire. (C. 1890–1.)

1896 BOX, CHARLES R., M.D., 2, Devonshire Place, Portland Place, W. *Trans.* 2.

1889 BOYD, STANLEY, B.S., 134, Harley Street, W. (C. 1899–1902.) *Trans.* 1, *C.C.* 7.

1890 BRADFORD, JOHN ROSE, M.D., F.R.S., 8, Manchester Square, W. (C. 1900–1902.) *Trans.* 2.

1878 BRIDGES, ROBERT, M.B., M.A., The Manor House, Yattendon, Berkshire. *Trans.* 1.

1868 BRIGHT, GEORGE CHARLES, M.D., Cannes, Alpes Maritimes, France.

O.M. BROADBENT, Sir WILLIAM HENRY, Bart., K.C.V.O., M.D., F.R.S., 84, Brook Street, W. (C. 1871–3, V.P. 1881–3, P. 1887–8.) *Trans.* 17.

1887 BROCK, J. H. E., M.D., B.S., 77, Fellows Road, South Hampstead, N.W.

1894 **BRODIE**, C. GORDON, Fernhill, Wootton Bridge, Isle of Wight. *C.C.* 1.

1898 **Brook**, WILLIAM HENRY BREFFIT, M.D., B.S., 33, Silver Street, Lincoln. *Trans.* 4, *C.C.* 1.

1890 BROWN, WALTER HENRY, 19, Queen Street, Leeds. *Trans.* 2.

ELECTED

1876 BROWNE, GEORGE BUCKSTON, 80, Wimpole Street, W. (C. 1891–3.) *Trans.* 3.

1904 BROWNE, H. S. D., M.B., B.C., 78, High Street, Winchester.

1893 BROWNE, JAMES WILLIAM, M.B., 37, Holland Park Avenue, W. *Trans.* 1.

1887 BROWNE, OSWALD AUCHINLECK, M.A., M.D., 7, Upper Wimpole Street, W.

1883 BRUCE, JOHN MITCHELL, M.D., 23, Harley Street, W. (C. 1901–4.)

1893 BRYANT, JOHN HENRY, M.D., 8, Mansfield Street, Portland Place, W. *Trans.* 2. *C.C.* 2.

O.M. BRYANT, THOMAS, M.Ch., 42, Norfolk Square, W. (C. 1872, V.P. 1876–7, P. 1885–6.) *Trans.* 9.

1890 BUCKLAND, FRANCIS O., B.A., M.B., C.M., Brockenhurst, Hants. *Trans.* 1.

1901 BUCKNALL, THOMAS RUPERT HAMPDEN, M.S., 16, Devonshire Street, W.

1891 BURGHARD, FREDERIC FRANÇOIS, M.D., M.S., 86, Harley Street, W.

1881 BURNET, ROBERT WILLIAM, M.D., 36, Grosvenor Street, W. *Trans.* 1.

1879 BURTON, WILLIAM EDWARD, 24, Wimpole Street, W.

1900 BUSCH, JOSEF PAUL ZUM, M.D., Chiswell House, 133, Finsbury Pavement, E.C. *Trans.* 1.

1881 BUTLIN, HENRY TRENTHAM, 82, Harley Street, W. (C. 1887–9, V.P. 1904- .) *Trans.* 8, *C.C.* 1.

1884 BUXTON, DUDLEY WILMOT, M.D., B.S., 82, Mortimer Street, W.

1902 BUZZARD, EDWARD FARQUHAR, M.D., 33, Harley Street, W. *Trans.* 1.

O.M. BUZZARD, THOMAS, M.D., 74, Grosvenor Street, W. (S. 1870–2, C. 1873–6, V.P. 1880–1, P. 1895–7.) *Trans.* 15, *C.C.* 3.

1901 CADDY, ADRIAN, M.B, 2/2, Harington Street, Calcutta.

1891 CADDY, ARNOLD, 2/2, Harington Street, Calcutta.

1893 CALEY, HENRY ALBERT, M.D., 24, Upper Berkeley Street, W. *C.C.* 1.

1890 CALVERT, JAMES, M.D., 113, Harley Street, Cavendish Square, W. *Trans.* 2, *C.C.* 2.

1893 CAMPBELL, HARRY, M.D., 23, Wimpole Street, W., and Coniston Cottage, Fox Lane, Upper Norwood, S.E. (C. 1904- .) *C.C.* 4.

1891 CARLESS, ALBERT, M.S., M.B., 10, Welbeck Street, W. (C. 1905– .)

1905 CARLING, E. ROCK, M.B., B.S., 38, New Cavendish Street, W.

1890 CARR, JOHN WALTER, M.D., 19, Cavendish Place, W. (C. 1903– .) *Trans.* 2, *C.C.* 6.

1901 CARSON, HERBERT WILLIAM, 26, Welbeck Street, Cavendish Square, W.

1901 Carwardine, THOMAS, M.S., 16, Victoria Square, Clifton, Bristol. *Trans.* 1.

1885 CATON, RICHARD, M.D., 86, Rodney Street, Liverpool. *Trans.* 3.

1890 CHAPMAN, CHARLES WILLIAM, M.D., 21, Weymouth Street, W. (C. 1900–3.) *Trans.* 2, *C.C.* 2.

1884 CHAPMAN, PAUL M., M.D., 1, St. John's Street, Hereford. *C.C.* 1.

ELECTED

1893 CHARSLEY, ROBERT STEPHEN, "The Old Barn," Slough.

1885 CHEYNE, W. WATSON, C.B., M.B., C.M., F.R.S., 75, Harley Street, W. (C. 1894–6.) *Trans.* 2, *C.C.* 8.

1873 CHISHOLM, EDWIN, M.D., 44, Roslyn Gardens, Darlinghurst, Sydney, New South Wales.

O.M. **CHURCH**, Sir WILLIAM SELBY, Bart., K.C.B., M.D., 130, Harley Street, W. (C. 1874–6.)

1873 CHURTON, THOMAS, M.D., 35, Park Square, Leeds. (C. 1889–91.) *Trans.* 6.

O.M. CLAPTON, EDWARD, M.D., Towercroft, 41, Eltham Road, Lee, S.E. (C. 1872–4.) *Trans.* 1.

1874 CLARK, ANDREW, 71, Harley Street, W. *Trans.* 1.

1887 CLARK, FRANCIS WILLIAM, Hong-Kong.

1898 CLARKE, JAMES JACKSON, M.B., 18, Portland Place, W. *Trans.* 2, *C.C.* 11.

1885 Clarke, J. MICHELL, M.D., 28, Pembroke Road, Clifton, Bristol.

1885 CLARKE, WILLIAM BRUCE, M.B., 51, Harley Street, W. (C. 1894–7.) *Trans.* 2, *C.C.* 3.

1877 Clay, ROBERT HOGARTH, M.D., 4, Windsor Villas, Plymouth.

1887 CLEMOW, ARTHUR HENRY WEISS, M.D., C.M., 101, Earl's Court Road, W. *Trans.* 1, *C.C.* 1.

1877 **CLUTTON**, HENRY HUGH, M.A. (*President*), 2, Portland Place, W. (C. 1885–7, V.-P. 1902–4, P. 1905– .) *Trans.* 12, *C.C.* 3.

1898 COBB, ERNEST HERBERT, Belmont, Stevenage, Herts.

1893 COLE, ROBERT HENRY, M.D., Moorcroft, Hillingdon, Middlesex.

1882 COLLIER, HERBERT, M.D., 21, South Quay, Great Yarmouth, Norfolk.

1902 COLLINS, VICTOR EVELYN, M.B., Graaff Reinet, Cape Colony.

1896 COLMAN, WALTER STACY, M.D., 9, Wimpole Street, W. (C. 1903– *Trans.* 2, *C.C.* 3.

1882 COLQUHOUN, DANIEL, M.D., Dunedin, New Zealand.

1868 COOPER, FRANK W. (address uncommunicated).

1900 COOPER, HARRY, M.D., Fownhope, Surbiton Hill, Surrey.

1903 COOPER, PERCY ROBERT, M.D., "Glenthorn," The Downs, Bowdon, Cheshire.

1902 CORNER, EDRED MOSS, B.Sc., M.B., 37, Harley Street, W. *Trans.* 4, *C.C.* 5.

1880 COTTLE, WYNDHAM, M.D., 39, Hertford Street, W.

1904 COTTON, HOLLAND JOHN, M.D., C.M., 33, Lowndes Street, S.W.

O.M. COUPER, JOHN, 80, Grosvenor Street, W. (C. 1874.)

1875 COUPLAND, SIDNEY, M.D., 16, Queen Anne Street, W. (S. 1882–4, C. 1885–7, V.-P. 1898–1901.) *Trans.* 5, *C.C.* 3.

1886 COUSINS, JOHN WARD, M.D., Riversdale, Kent Road, Southsea. *Trans.* 1.

1882 COXWELL, C. F., M.B., Brighton, Melbourne, Victoria. *Trans.* 2.

1897 CRAWFURD, RAYMOND H. P., M.D., 71, Harley Street, Cavendish Square, W. *C.C.* 3.

ELECTED

1879 CRIPPS, WILLIAM HARRISON, 2, Stratford Place, W. (C. 1886–8.) *Trans.* 3.

1872 CRITCHETT, Sir G. ANDERSON, 21, Harley Street, W.

1877 CROCKER, HENRY RADCLIFFE, M.D., 121, Harley Street, W. (C. 1884–5.) *Trans.* 16.

1896 CROOKE-LAWLESS, W. R., Surg.-Maj. A.M.S., M.D., Guards' Club, S.W.

1898 CROUCH, HERBERT CHALLICE, 31, Welbeck Street, Cavendish Square, W.

1905 CUNNING, JOSEPH, M.B., B.S., 77, Wimpole Street, W.

1893 CURTIS, HENRY JONES, M.D., B.S., Memorial Hospital, Buluwayo, Rhodesia. *C.C.* 1.

1902 CUTHBERT, CHARLES FIRMIN, 2, Barton Street, Gloucester.

1872 DALBY, Sir WILLIAM BARTLETT, M.B., 18, Savile Row, W. (C. 1879–81.) *Trans.* 4.

1896 DALTON, NORMAN, M.D., 4, Mansfield Street, Portland Place, W. (C. 1904.) *Trans.* 4, *C.C.* 7.

1898 DARLEY-HARTLEY, WILLIAM, M.D., Seapoints, Cape Town, South Africa.

1893 DAUBER, JOHN HENRY, M.B., B.Ch., 29, Charles Street, Berkeley Square, W.

1886 DAVIES, ARTHUR T., M.D., 23, Finsbury Square, E.C. (C. 1897–1900.) *C.C.* 13.

1879 Davy, HENRY, M.D., 29, Southernhay, Exeter.

1889 DEAN, HENRY PERCY, M.B., 69, Harley Street, W.

1897 DEANE, JOHN HENRY, 50, Wallwood Road, Leytonstone, Essex.

1899 DE CHAZAL, EDMOND LUCIEN, M.D., Port Louis, Mauritius.

1903 DELBRUCK, RAOUL ERNEST, M.B., 13, Buckingham Gate, S.W.

1879 Dennis, FREDERIC S., M.D., 542, Madison Avenue, New York, U.S.A.

1875 DENT, CLINTON T., 61, Brook Street, W. (C. 1884–6, V.P. 1905– .) *Trans.* 5. *C.C.* 1.

O.M. DICKINSON, WILLIAM HOWSHIP, M.D., 10, Stanhope Place, Marble Arch, W. (C. 1874–5, V.P. 1888–9.) *Trans.* 2.

1894 Dickson, THOMAS HUGH, M.B., B.C., H.M. Customs, Lower Thames Street, E.C.

1871 DIVER, EBENEZER, M.D., 7, Pittville Terrace, Stamshaw Road, Portsmouth. (C. 1890–2.) *Trans.* 1.

1901 DOUGLAS, JOHN J., M.D., 42, Central Hill, Norwood, S.E.

1868 DRAGE, CHARLES, M.D., Hatfield, Herts.

1896 DREW, DOUGLAS, M.D., B.S., 49, Devonshire Street, Portland Place, W. *Trans.* 1. *C.C.* 1.

O.M. DUCKWORTH, Sir DYCE, M.D., LL.D. 11, Grafton Street, Bond Street, W. (C. 1875–7, V.P. 1887–8, P. 1891–2.) *Trans.* 24, *C.C.* 8.

1902 DUDGEON, LEONARD S., 6, Powis Gardens, Bayswater, W. *Trans.* 1, *C.C.* 2.

1884 DUKE, EDGAR, 11, Wilbury Road, Hove.

1869 DUKE, OLLIVER THOMAS, M.B., Surgeon, Bengal Army, India.

ELECTED

1899 DUNCAN, ANDREW, M.D., 24, Chester Street, Grosvenor Place, S.W. *Trans.* 1.

1889 DUNCAN, JOHN, M.D., St. Petersburg.

1889 DUNN, LOUIS ALBERT, M.S., 51, Devonshire Street, Portland Place, W. (C. 1899–1902.) *Trans.* 2, *C.C.* 2.

1884 EDMUNDS, WALTER, M. C., 2, Devonshire Place, Portland Place, W. (C. 1902–5.) *Trans.* 2.

1899 EID, ALFRED, M.D., Cairo, Egypt.

1902 ELLIOT, ANDREW, M.D., 11, Upper Berkeley Street, Portman Square, W.

1882 EMOND, EMILE, M.D., 113, Boulevard Beaumarchais, Paris.

1902 ENGLISH, THOMAS CRISP, B.S., 31, Grosvenor Street, W. *C.C.* 1.

1893 EVANS, WILLMOTT H., M.D., B.S., 2, Upper Wimpole Street, Cavendish Square, W. *C.C.* 1.

1888 **EVE**, FREDERIC S., 61, Harley Street, Cavendish Square, W. (C. 1899–1902.) *Trans.* 5, *C.C.* 10.

1877 **EWART**, WILLIAM, M.D., 33, Curzon Street, W. (C. 1884–6, V.P. 1905– .) *Trans.* 5, *C.C.* 4.

1899 FAGGE, CHARLES HERBERT, M.S., 22, St. Thomas's Street, London Bridge, S.E. *Trans.* 1, *C.C.* 1.

1868 **Fairbank**, FREDERICK ROYSTON, M.D., Westcott, Dorking, Surrey. *Trans.* 2.

1905 FAIRBANK, HAROLD A. T., 137, Harley Street, W. *C.C.* 1.

1889 FARDON, EDWARD ASHBY, Middlesex Hospital, W.

1900 FAWCETT, JOHN, M.D., 66, Wimpole Street, W. *Trans.* 2, *C.C.* 2.

1902 FEDDEN, WALTER FEDDE, M.B., M.S., 20, Lower Seymour Street, Portman Square, W.

1902 FENNELL, CHARLES HENRY, M.D., County Asylum, Hellingly, Sussex. *C.C.* 1.

1887 FENWICK, E. HURRY, 14, Savile Row, W. *Trans.* 2, *C.C.* 2.

1872 FENWICK, J. C. J., M.D., Long Framlington, Morpeth.

1893 FENWICK, WILLIAM SOLTAU, M.D., 29, Harley Street, W. *Trans.* 2.

1878 FIELD, GEORGE P., 34, Wimpole Street, W.

1876 FINLAY, Prof. DAVID WHITE, M.D., 2, Queen's Terrace, Aberdeen. (C. 1885–7, S. 1891.) *Trans.* 5.

1894 FLETCHER, HERBERT MORLEY, M.D., 98, Harley Street, W. *Trans.* 1, *C.C.* 1.

1878 **FONMARTIN**, HENRY DE, M.D. (Travelling.)

1905 FORSYTH, DAVID, M.D., 6, St. Thomas's Street, London Bridge, S.E.

1890 FOSTER, MICHAEL G., M.D., M.A., Villa San Giovanni, San Remo, Italy.

1899 FOWLER, CHARLES OWEN, M.D., Cotford House, Thornton Heath, Surrey.

1881 FOWLER, JAMES KINGSTON, M.D., 35, Clarges Street, W. (C. 1887–9, 1899–1902, S. 1896–9, V.-P. 1904– .) *Trans.* 4, *C.C.* 6.

1886 FOX, R. HINGSTON, M.D., 29, Weymouth Street, Portland Place, W. *C.C.* 1.

ELECTED

1878 FOX, THOMAS COLCOTT, M.B., 14, Harley Street, W. (C. 1888–90.) *Trans.* 7, *C.C.* 2.

1893 FOXWELL, ARTHUR, M.D., 22, Newhall Street, Birmingham.

1902 FRENCH, HERBERT, M.D., 26, St. Thomas's Street, London Bridge, S.E. *C.C.* 1.

1897 FRIPP, Sir ALFRED DOWNING, C.B., M.V.O., M.S., 19, Portland Place, W. (C. 1904– .) *Trans.* 3.

1899 FÜRTH, KARL, M.D., 39, Harley Street, W.

1890 FULLER, HENRY ROXBURGH, M.D., 45, Curzon Street, W.

1897 FURNIVALL, PERCY, 28, Weymouth Street, Portland Place, W.

1891 FYFFE, W. KINGTON, M.B., B.C., 19, Duke Street, Manchester Square, W. (Travelling, New Zealand.) *Trans.* 1.

1888 GAGE-BROWN, CHARLES HERBERT, M.D., 85, Cadogan Place, S.W.

1895 GALLOWAY, JAMES, M.D., 54, Harley Street, W.

1887 GARROD, ARCHIBALD EDWARD, M.A., M.D. (*Hon. Secretary*), 9, Chandos Street, Cavendish Square, W. (C. 1898–1901, S. 1905– .) *Trans.* 4, *C.C.* 4.

1879 GARSTANG, THOMAS WALTER HARROPP, Edge Mount, Altrincham.

1903 GATES, EDWARD A., via Palestro A., Florence.

1885 GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan Place, S.W. *Trans.* 1.

1893 GIBBS, CHARLES, 115, Harley Street, W. *C.C.* 1.

1868 GLOVER, JAMES GREY, M.D., 25, Highbury Place, N. (C. 1878–80. V.P. 1892–4.) *Trans.* 2.

1893 GLOVER, LEWIS G., M.B., B.C., 17, Belsize Park, Hampstead, N.W.

1875 GODLEE, RICKMAN JOHN, M.S., 19, Wimpole Street, W. (C. 1882–3, 1887–9, S. 1884–6, V.P. 1895–8.) *Trans.* 14, *C.C.* 10.

1901 GOFFE, ERNEST GEORGE LEOPOLD, M.D., Western Hospital, Seagrave Road, Fulham, S.W.

1882 GOLDIE, ROBERT WILLIAM, Hirst House, Hirst, Morpeth, Northumberland.

1878 **GOLDING-BIRD**, C. H., M.B., 12, Queen Anne Street, W. (C. 1887–9, V.-P. 1903–5.) *Trans.* 18, *C.C.* 1.

1894 GOODALL, EDWARD WILBERFORCE, M.D., Eastern Hospital, Homerton, N.E. (C. 1904– .) *Trans.* 7.

1875 GOODHART, JAMES FREDERIC, M.D., 25, Portland Place, W. (C. 1880–2, V.P. 1897–1900.) *Trans.* 14, *C.C.* 2.

1891 GOODMAN, ROGER NEVILLE, M.B., Elmside, Kingston-on-Thames.

1869 GOODRIDGE, HENRY FREDERICK AUGUSTUS, M.D., 10, Brock Street, Bath.

1882 GOODSALL, D. H., 17, Devonshire Place, W.

1877 GOULD, A. PEARCE, M.S., 10, Queen Anne Street, W. (C. 1885–7, 1895–8, S. 1892–5, V.-P. 1898–1901.) *Trans.* 14, *C.C.* 9.

1901 GOWAN, BOWIE CAMPBELL, Raven Dene, Great Stanmore, Middlesex.

ELECTED

1875 GOWERS, Sir WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne Street, W. (C. 1881–2, V.-P. 1903–5.) *Trans.* 5, *C.C.* 1.

1904 GRAHAM, GEORGE HERBERT, M.D., 14, Old Cavendish Street, W.

1891 GRANT, J. DUNDAS, M.D., 18, Cavendish Square, W.

1868 GREEN, T. HENRY, M.D., 74, Wimpole Street, W. (C. 1877–9.) *Trans.* 2, *C.C.* 1.

1875 Greenfield, WILLIAM SMITH, M.D., 7, Heriot Row, Edinburgh. (C. 1881.) *Trans.* 3.

1893 GRIFFITH, WALTER SPENCER ANDERSON, M.D., 96, Harley Street, W.

1895 Grube, KARL, M.D., Neuenahr, Germany. *Trans.* 1.

1895 GUTHRIE, LEONARD GEORGE, M.D., 15, Upper Berkeley Street, W. *Trans.* 1, *C.C.* 7.

1887 HABERSHON, SAMUEL HERBERT, M.D., 86, Harley Street, W. (C. 1901–4.) *C.C.* 1.

1900 HADLEY, WILFRED J., M.D., 33, Queen Anne Street, Cavendish Square, W.

1875 HALE, C. D. B., 3, Sussex Place, W. *Trans.* 1.

1903 HALL, JOHN BASIL, M.C., 31, Manningham Lane, Bradford.

1878 HALL, F. DE HAVILLAND, M.D., 47, Wimpole Street, W. (C. 1885–7, V.-P. 1904– .) *Trans.* 8, *C.C.* 5.

1889 HALSTEAD, GEORGE EZRA, M.D., B.S., Albion Hill House, Ramsgate.

1888 HANDFIELD-JONES, MONTAGU, M.D., 35, Cavendish Square, W.

1886 Handford, HENRY, M.D., 6, Regent Street, Nottingham. (C. 1893–4.) *Trans.* 8, *C.C.* 1.

1904 HARBURN, JOHN ENGLISH, 8, Broad Walk, Buxton, Derbyshire.

1886 HARDIE, JAMES, M.D., 15, St. John Street, Manchester.

1890 HARPER, JAMES, M.D., 25, Rosary Gardens, South Kensington, S.W.

1872 HARRIS, HENRY, M.D., Trengweath, Redruth, Cornwall.

1889 HARRIS, HERBERT ELWIN, M.B., 13, Lansdown Place, Victoria Square, Clifton, Bristol. *Trans.* 1.

1903 HARRIS, WILFRED, M.D., 61, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 2.

1881 HARRISON, CHARLES EDWARD, M.B., Grenadier Guards Hospital, Rochester Row, S.W.

1892 HARRISON, DAMER, 53, Rodney Street, Liverpool. *Trans.* 1.

1869 HAWARD, J. WARRINGTON, 57, Green Street, Grosvenor Square, W. (C. 1876–8, 1884–6, S. 1881–3, V.P. 1888.) *Trans.* 15.

1899 Hawkes, CLAUDE SOMERVILLE, Glencairn, Wickham Terrace, Brisbane, Queensland, Australia.

1886 HAWKINS, FRANCIS HENRY, M.D., 73, London Street, Reading. (C. 1902–5.) *Trans.* 4, *C.C.* 3.

1889 HAWKINS, HERBERT PENNELL, M.D., B.Ch., 56, Portland Place, W. (C. 1900–3.) *Trans.* 1.

ELECTED

1890 HAWKINS-AMBLER, GEORGE ARTHUR, 30, Rodney Street, Liverpool.

1900 HAYWARD, JOHN ARTHUR, M.D., 23, The Grange, Wimbledon Common, S.W.

1903 HEATH, PHILIP MAYNARD, M.B., 36, Cavendish Square, W.

1879 HENDERSON, GEORGE COURTENAY, M.D., Kingston, Jamaica, West Indies.

1884 HERRINGHAM, WILMOT PARKER, M.D., 40, Wimpole Street, W. (C. 1894–7.) *Trans.* 6. *C.C.* 3.

1897 HILLIER, ALFRED P., M.D., C.M., 30, Wimpole Street, W.

1874 HOLDERNESS, WILLIAM BROWN, 15, Park Street, Windsor.

1868 **Holman**, Sir CONSTANTINE, M.D., 26, Gloucester Place, Portman Square, W. (C. 1894–7.)

1878 HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green Street, Park Lane, W. (C. 1891–3.) *Trans.* 1.

1884 HORSLEY, Sir VICTOR, M.B., F.R.S., 25, Cavendish Square, W. (C. 1894.) *Trans.* 2, *C.C.* 1.

1901 HORTON-SMITH-HARTLEY, PERCIVAL, M.D., 19, Devonshire Street, Portland Place, W.

1895 HOUGH, CHARLES HENRY, The Cottage, Clappersgate, Ambleside, Windermere.

1880 **HOVELL**, T. MARK, 105, Harley Street, W. *Trans.* 1.

1893 HOWARD, R. J. BLISS, M.D., 31, Queen Anne Street, W.

1898 HULKE, SYDNEY BACKHOUSE, Ivy House, Walmer, Kent.

1897 HUMPHRYS, CHARLES B., Eagle House, Blandford, Dorset.

1897 HUNT, GEORGE BERTRAM, M.B., 47, Albemarle Crescent, Scarborough. *Trans.* 1, *C.C.* 1.

1892 HUNTER, WILLIAM, M.D., 103, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.S., 15, Cavendish Square, W. (C. 1867–8, V.P. 1875–6.) *Trans.* 14, *C.C.* 1.

1896 HUTCHINSON, JONATHAN, jun., 15, Cavendish Square, W. (C. 1902–5.) *Trans.* 6, *C.C.* 8.

1893 IONIDES, THEODORE HENRY, M.B., B.S., 25, First Avenue, Brighton.

1883 JACKSON, GEORGE HENRY, Ashburton, Carew Road, Eastbourne.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.S., 3, Manchester Square, W. (C. 1872–3, V.P. 1887.) *Trans.* 2, *C.C.* 1.

1888 JAMES, JAMES THOMAS, M.D., 30, Harley Street, W.

1901 JOHNSON, EDWARD ANGAS, M.B., B.S., St. Catherine's, Prospect, South Australia.

1889 JOHNSON, RAYMOND, M.B., B.S., 11, Wimpole Street, W. (C. 1900–3.) *Trans.* 6, *C.C.* 6.

1893 JOHNSTON, G. F., M.D., 3, Montagu Place, Montagu Square, W.

ELECTED

1904 JONES, A. ERNEST, M.D., B.Sc., 13, Harley Street, W.
1903 JONES, ERNEST LLOYD, 59, Trumpington Street, Cambridge.
1902 JONES, LAWRENCE, 3, The Terrace, Champion Hill, S.E.
1898 Jones, ROBERT, 11, Nelson Street, Liverpool. *Trans.* 2.
1872 JONES, THOMAS RIDGE, M.D., 4, Chesham Place, S.W. (C. 1892–3.)
1876 JORDAN, FURNEAUX, 10, Ferndale, Teignmouth, S. Devon. *Trans.* 1.
1902 JOWERS, REGINALD F., 55, Brunswick Square, Brighton.
1878 KEETLEY, CHARLES ROBERT BELL, 56, Grosvenor Street, W. (C. 1895–8.) *Trans.* 2.
1898 KELLOCK, THOMAS HERBERT, 8, Queen Anne Street, Cavendish Square, W. (C. 1903– .) *Trans.* 3, *C.C.* 6.
1901 KELYNACK, THEO. N., M.D., 120, Harley Street, W.
1901 KEOGH, Lieut.-Col. ALFRED, C.B., M.D., 18, Victoria Street, S.W.
1897 KEYSER, CHARLES RALPH, 29, Hamilton Terrace, N.W. *Trans.* 1, *C.C.* 5.
1883 KIDD, PERCY, M.D., 60, Brook Street, W. (C. 1891–2, 1902–5, S. 1899–1902.) *Trans.* 5, *C.C.* 10.
1887 Knaggs, R. LAWFORD, B.C., 27, Park Square, Leeds. *Trans.* 2.
1878 LACEY, THOMAS WARNER, 196, Burrage Road, Plumstead.
1897 LAMPLOUGH, CHARLES, Kirkstall, Alverstoke, Hants.
1890 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Winchester House. Swansea, S. Wales. *Trans.* 2.
1895 LANE, JAMES ERNEST, 46, Queen Anne Street, W. C. 1905– . *Trans.* 1.
1883 LANE, WILLIAM ARBUTHNOT, M.B., M.S., 21, Cavendish Square, W. (C. 1893–6.) *Trans.* 29, *C.C.* 10.
O.M. **LANGTON**, JOHN, 62, Harley Street, W. (C. 1878–80, V.P. 1892–4, P. 1897–9.) *Trans.* 5.
1886 LANKESTER, HERBERT, M.D., Ingleborough, Woking, Surrey.
1885 LARDER, HERBERT, Whitechapel Infirmary, Vallance Road, E. *C.C.* 1.
1893 LAWSON, ARNOLD, M.D., 12, Harley Street, W.
1900 LEAF, CECIL H., M.B., 75, Wimpole Street, Cavendish Square, W.
1877 Lediard, HENRY AMBROSE, M.D., 35, Lowther Street, Carlisle. (C. 1889, V.-P. 1902–4.) *Trans.* 5, *C.C.* 1.
1896 Leech, PRIESTLEY, M.D., King Cross, Halifax.
1877 LEES, DAVID B., M.D., 22, Weymouth Street, W. (C. 1885, V.-P. 1902–4.) *Trans.* 6.
1902 LEGG, THOMAS PERCY, M.B., 141, Harley Street, Cavendish Square, W.
1899 LESLIE, R. MURRAY, M.D., 26, Harley Street, Cavendish Square, W. *C.C.* 1.
1901 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., 72, Harley Street, Cavendish Square, W.
1892 LEWIS, EDWARD JOHN, M.B., B.C., 74, Hamilton Terrace, N.W.
1895 LEWIS, ERNEST E., M.D., 30, Weymouth Street, W.
1903 LITTLE, E. GRAHAM, M.D., 61, Wimpole Street, W.

ELECTED

1890 LITTLE, JOHN FLETCHER, M.B., 32, Harley Street, W. *C.C.* 5.

1891 LITTLEWOOD, HARRY, 25, Park Square, Leeds. *Trans.* 1.

1875 LIVEING, EDWARD, M.D., 52, Queen Anne Street, W.

1885 LOCKWOOD, CHARLES BARRETT, 19, Upper Berkeley Street, W. (C. 1896–9.) *Trans.* 7.

1876 LONGHURST, ARTHUR EDWIN TEMPLE, M.D., 26, Lower Belgrave Street, Eaton Square, S.W. (C. 1889–91.) *Trans.* 1.

1904 LONGHURST, FREDERIC W., 26, Lower Belgrave Street, Eaton Square, S.W.

1897 LOW, HAROLD, M.B., 10, Evelyn Gardens, South Kensington, S.W.

1899 LOW, V. WARREN, M.D., B.S., 27, Queen Anne Street, Cavendish Square, W. *Trans.* 1.

1881 LUBBOCK, MONTAGU, M.D., 19, Grosvenor Street, W.

1876 LUCAS, R. CLEMENT, M.B., B.S., 50, Wimpole Street, W. (C. 1883–5, V.P. 1901–3.) *Trans.* 12. *C.C.* 1.

1894 LUFF, ARTHUR PEARSON, M.D., 9, Queen Anne Street, Cavendish Square, W.

1879 LUNN, JOHN REUBEN, St. Marylebone Infirmary, Rackham Street, Ladbroke Grove Road, W. (C. 1890–1, V.P. 1905– .) *Trans.* 8, *C.C.* 20.

1893 LYS, HENRY GRABHAM, M.D., Southbrook, Suffolk Road, Bournemouth.

1889 MACBRIDE, P., M.D., 16, Chester Street, Edinburgh.

1891 MACDONALD, GRENVILLE, M.D., 85, Harley Street, W.

1901 MCGAVIN, LAURIE HUGH, 6, Mansfield Street, Cavendish Square, W. *Trans.* 2, *C.C.* 3.

1881 MCHARDY, MALCOLM MACDONALD, 5, Savile Row, W. *Trans.* 1.

1891 MACKENZIE, HECTOR W. G., M.A., M.D., 34, Upper Brook Street, W. (C. 1902–5.) *Trans.* 6, *C.C.* 1.

1879 MACKENZIE, SIR STEPHEN, M.D., 18, Cavendish Square, and Merry-court, Gt. Bookham, Leatherhead. (C. 1884, 1888–90, S. 1885–7. V.P. 1900–2.) *Trans.* 9, *C.C.* 10.

1884 MACKERN, JOHN, M.B., St. German's Lodge, Shooter's Hill Road, Blackheath, S.E.

1885 MACLAREN, RODERICK, M.D., Portland Square, Carlisle. *Trans.* 1.

1898 MACREADY, JONATHAN F.C. H., 42, Devonshire Street, Portland Place, W.

1879 MAGILL, JAMES, M.D., M.C., Coldstream Guards, Queen Anne's Mansions, S.W.

1885 MAGUIRE, ROBERT, M.D., 4, Seymour Street, W. *Trans.* 1.

1881 MAKINS, GEORGE HENRY, C.B. (*Treasurer*), 47, Charles Street, Berkeley Square, W. (C. 1892–4, 1898–1901, S. 1895–8, T. 1903– .) *Trans.* 5, *C.C.* 7.

1887 MALCOLM, JOHN D., M.B., C.M., 13, Portman Street, W. (C. 1900–3.) *Trans.* 1, *C.C.* 2.

ELECTED

1890 MANSON, Sir PATRICK, K.C.M.G., M.D., C.M., F.R.S., 21, Queen Anne Street, W. (C. 1895–7.) *C.C.* 1.

1902 MARRIOTT, CECIL, M.B., M.C., 11, Welford Road, Leicester.

1888 Marriott, HYDE, M.B., Dial House, Stockport.

1868 MARSH, Professor F. HOWARD, 14, Hertford Street, Mayfair, W. (C. 1876–7. 1881–3, S. 1878–80, V.P. 1887–8, T. 1899–1901, P. 1901–3.) *Trans.* 20, *C.C.* 7.

1887 MARTIN, SIDNEY, M.D., B.S., F.R.S., 10, Mansfield Street, W. (C. 1896–9.)

1888 MASON, DAVID JAMES, M.D., C.M., Rosemont, Maidenhead.

1892 MASTERS, JOHN ALFRED, M.D., 94, Knightsbridge, S.W.

1884 MAUDSLEY, HENRY CARE, M.D., 22, Collins Street, Melbourne, Victoria

1897 MAY, CHICHESTER GOULD, M.D., 59, Cadogan Place, S.W.

1868 May, EDWARD HOOPER, M.D., High Cross, Tottenham, Middlesex.

1888 MAY, WILLIAM PAGE, M.D., B.Sc., 9, Manchester Square, W.; and Helouan, nr. Cairo, Egypt (October to April).

1901 MENZIES, F. U., Edinburgh University Union, Edinburgh.

1888 MENZIES, J. HERBERT, 47, Earl's Court Square, S.W.

1893 MERCER, WILLIAM BRACEWELL, M.B., B.C., Cliffe Cottage, Ripponden, near Halifax, Yorks.

1878 MEREDITH, WILLIAM APPLETON, C.M., 21, Manchester Square, W. (C. 1887–9.) *Trans.* 2.

1894 MICHELS, ERNST, M.D., 48, Finsbury Square, E.C.

1873 MICKLE, WILLIAM JULIUS, M.D., Grove Hall Asylum, Bow, E. (C. 1897–1900.)

1890 MILEY, MILES, M.A., M.B., 21, Belsize Avenue, Hampstead, N.W.

1896 MILLS-ROBERTS, ROBERT HERBERT, C.M.G., Llanberis, North Wales. *Trans.* 1.

1902 MOON, R. O., M.D., 50, Green Street, Grosvenor Square, W.

1874 MORGAN, JOHN HAMMOND, C.V.O., 68, Grosvenor Street, W. (C. 1883–5, V.P. 1898–1901.) *Trans.* 2, *C.C.* 8.

1888 MORISON, ALEXANDER, M.D., 14, Upper Berkeley Street, W. *Trans.* 2, *C.C.* 1.

1899 MORISON, RUTHERFORD, 14, Saville Row, Newcastle-on-Tyne. *Trans.* 1.

1903 MORLEY, ARTHUR S., 29, Gower Street, W.C.

1877 MORRIS, HENRY, M.A., M.B., 8, Cavendish Square, W. (C. 1884–6, V.P. 1903–5.) *Trans.* 9.

1877 MORRIS, MALCOLM ALEX., 8, Harley Street, W. (C. 1890–2.) *Trans.* 1, *C.C.* 1.

1901 MORSE, THOMAS HENRY, All Saints' Green, Norwich. *Trans.* 1.

1902 MORTON, CHARLES ALFRED, 14, Vyvyan Terrace, Clifton, Bristol. *Trans.* 1.

1885 MOTT, FREDERICK WALKER, M.D., F.R.S., 25, Nottingham Place, W. (C. 1895–8.) *Trans.* 1, *C.C.* 4.

1899 MOUAT-BIGGS, CHARLES E. FORBES, 2, Fauconberg Villas, Bayshill, Cheltenham. *C.C.* 2.

ELECTED

1879 MOULLIN, CHARLES W. MANSELL, 69, Wimpole Street, W. (C. 1888–90.) *Trans.* 11, *C.C.* 4.

1900 MOYNIHAN, BERKELEY GEORGE ANDREW, M.S., 33, Park Square, Leeds. (C. 1904– .) *Trans.* 2.

1875 MURPHY, Sir SHIRLEY F., 9, Bentinck Terrace, Regent's Park, N.W. (C. 1888–90.) *C.C.* 1.

1885 MURRAY, ALEXANDER DALTON, M.B., Colombo, Ceylon.

1893 MURRAY, GEORGE REDMAYNE, M.D., 11, Ellison Place, Newcastle-on-Tyne.

1883 MURRAY, HUBERT MONTAGUE, M.D., 25, Manchester Square, W. (C. 1893–6.) *Trans.* 1.

1894 MURRAY, JOHN, 110, Harley Street, W. *C.C.* 1.

1872 MYRTLE, ANDREW S., M.D., 10, Park Parade, Harrogate. (C. 1892.)

1902 NABARRO, DAVID, M.D., 6, Crown Office Row, Inner Temple, E.C.

1892 NASH, WALTER GIFFORD, 31, St. Peter's, Bedford.

1889 Newman, D., M.D., 28, Woodside Place, Glasgow. *Trans.* 4.

O.M. NORTON, ARTHUR TREHERN, C.B., Leyfields Wood, Ashampstead, Berks. (C. 1874–6.) *Trans.* 7.

1880 O'CONNOR, BERNARD, M.D., Twyford Abbey, Willesden, N.W. *Trans.* 1.

1899 OGLE, CYRIL, M.B., 96, Gloucester Place, Portman Square, W. *Trans.* 1.

1887 Oliver, THOMAS, M.D., 7, Ellison Place, Newcastle-upon-Tyne.

1887 OPENSHAW, THOMAS HORROCKS, C.M.G., M.B., 16, Wimpole Street, W. (C. 1905– .) *Trans.* 1, *C.C.* 5.

1868 OPPERT, FRANZ, M.D., 128, Leipzigerstrasse, Friedenau, Germany. *Trans.* 1.

1889 ORD, W. WALLIS, M.D., B.Ch., The Hall, Salisbury. *Trans.* 1, *C.C.* 1.

1887 ORMEROD, JOSEPH ARDERNE, M.D., 25, Upper Wimpole Street, W. (C. 1899–1902.) *C.C.* 2.

1884 ORMSBY, Sir LAMBERT HEPENSTAL, M.D., 92, Merrion Square West, Dublin.

1888 OXLEY, ALFRED RICE, M.D., 7, Courtfield Road, South Kensington, S.W.

1888 PAGE, FREDERICK, M.D., 1, Saville Place, Newcastle-on-Tyne.

1875 PAGE, HERBERT W., M.C., M.B., 146, Harley Street, W. (C. 1882–4, V.P. 1899–1902.) *Trans.* 8, *C.C.* 2.

1884 PAGET, STEPHEN, 70, Harley Street, W. (C. 1898–1901.) *Trans.* 9, *C.C.* 3.

1881 PARKER, RUSHTON, M.B., B.S., 59, Rodney Street, Liverpool. (C. 1896–9.) *Trans.* 1, *C.C.* 2.

1890 PARKIN, ALFRED, M.S., 24, Albion Street, Hull. *Trans.* 2.

1894 PARKINSON, JOHN PORTER, M.D., 57, Wimpole street, W. *Trans.* 1, *C.C.* 5.

ELECTED

1898 PARTRIDGE, ARTHUR A. H., M.D., M.Ch., St. Germans, Sutton, Surrey.

1881 PASTEUR, WILLIAM, M.D., 4, Chandos Street, W. (C. 1891–3, 1905– , S. 1902–5.) *Trans.* 7, *C.C.* 5.

1893 PATERSON, DONALD ROSE, M.D., C.M., 15, St. Andrew's Crescent, Cardiff.

1900 PATON, EDWARD PERCY, M.S., 53, Queen Anne Street, Cavendish Square, W. *Trans.* 2.

1892 PAUL, FRANK THOMAS, 38, Rodney Street, Liverpool. *Trans.* 1.

1893 PAUL, JOHN ERNEST, M.B., 26, Queensborough Terrace, Kensington Gardens, W. *Trans.* 1.

O.M. PAVY, FREDERICK WILLIAM, M.D., F.R.S., 35, Grosvenor Street, W. (C. 1869–71, V.P. 1882–4.) *Trans.* 3.

1902 PEARSON, S. VERE, M.B., B.C., Woburn Lodge, Broughton Park, Manchester. *C.C.* 2.

1879 PEEL, ROBERT, 120, Collins Street East, Melbourne, Victoria.

1887 PENROSE, FRANCIS GEORGE, M.D., 84, Wimpole Street, W. (C. 1896–9.) *Trans.* 1, *C.C.* 2.

1882 PEPPER, AUGUSTUS JOSEPH, M.S., M.B., 13, Wimpole Street, W. *Trans.* 1.

1900 PERKINS, GEORGE STEELE, M.D., 85, Wimpole Street, W.

1898 PERKINS, J. J., M.D., 41, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1895 PHEAR, ARTHUR G., M.D., B.C., 47, Weymouth Street, Portland Place, W. *Trans.* 2.

1895 PHILLIPS, F. B. WILLMER, M.B., 7, Harper Place, Bedford.

1885 PHILLIPS, SIDNEY, M.D., 3, Upper Brook Street, W. (C. 1893–6.) *Trans.* 10, *C.C.* 6.

1885 PITT, GEORGE NEWTON, M.D., 15, Portland Place, W. (C. 1894–6.) *Trans.* 7, *C.C.* 6.

1883 PITTS, BERNARD, M.A., M.C., 109, Harley Street, W. (C. 1893.) *Trans.* 5.

1871 Playne, ALFRED, M.B., Maidenhead.

1884 POLLARD, BILTON, B.S., 24, Harley Street, W. (C. 1895–8.) *Trans.* 2.

1868 POLLOCK, JAMES EDWARD, M.D., 37, Collingham Place, S.W. (C. 1878–80.)

1881 POWELL, H. A., M.A., 44, Sandgate Road, Folkestone.

O.M. POWELL, Sir R. DOUGLAS, Bart., K.C.V.O., M.D., 62, Wimpole Street, W. (C. 1874–6, V.P. 1889–90, P. 1899-1901.) *Trans.* 4.

1868 PRENTIS, CHARLES, Surgeon-Major, Bengal Medical Service; India.

1902 PRICE, F. W., M.B., 77, Wimpole Street, W. (C. 1904.)

1884 PRINGLE, JOHN JAMES, M.B., 23, Lower Seymour Street, W. (C. 1890–2, 1897–1900.) *Trans.* 1, *C.C.* 3.

1884 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., 48, Brook Street, W. (C. 1890–2.) *Trans.* 1.

ELECTED

1896 PYE-SMITH, RUTHERFORD JOHN, 350, Glossop Road, Sheffield.
1893 RAKE, ALFRED THEODORE, M.B., B.S., 58, Delancey Street, Regent's Park, N.W.
1895 RAMSAY, HERBERT MURRAY, 35A, Hertford Street, Mayfair, W.
1889 RANKING, JOHN E., M.D., Hanover House, Tunbridge Wells.
1883 READ, THOMAS LAURENCE, 11, Petersham Terrace, Queen's Gate, S.W.
1902 REID, Sir JAMES, Bart., G.C.V.O., K.C.B., 72, Grosvenor Street, Grosvenor Square, W.
1901 REISSMANN, CHARLES, M.D., College Town, Adelaide, South Australia.
1899 Rennie, GEORGE EDWARD, M.D., College Street, Hyde Park, Sydney, N.S.W.
1868 RICK, MICHAEL W., M.D., Elmbank, Hargrave, Northamptonshire, (C. 1876–8.)
1898 RICHARDS, JOSEPH STEWART, M.D., B.S., 5, Wellesley Villas, Wellesley Road, Croydon, S.E. *C.C.* 2.
1903 RIGBY, HUGH, M.S., 7, Wimpole Street, Cavendish Square, W.
O.M. RINGER, SYDNEY, M.D., F.R.S., 15, Cavendish Place, W. (C. 1871–2.) *Trans.* 1.
1896 RISDON, WILLIAM ELLIOT, M.D., 4, Etchingham Park Road, Church End, Finchley, N.
1903 RIVIERE, CLIVE, M.D., 19, Devonshire Street, W. *C.C.* 1.
1873 Roberts, DAVID LLOYD, M.D., 11, St. John Street, Manchester.
1888 ROBERTS, FRANK ERNEST, Tulse Dale Villa, Lower Norwood, S.E.
1883 ROBERTS, FREDERICK THOMAS, M.D., 102, Harley Street, W. (C. 1892–4.)
1900 ROBERTS, GRIFFITH LLOYD, M.B., C.M., Castle Square, Carnarvon, North Wales.
1890 ROBERTSON, ROBERT, M.D., Belgrave Road, Ventnor, Isle of Wight.
1885 ROBINSON, ARTHUR HENRY, M.D., St. Mary's Infirmary, Highgate Hill, N. *C.C.* 3.
1892 ROBINSON, HENRY BETHAM, M.D., M.S., 1, Upper Wimpole Street, W. (C. 1901–4). *Trans.* 10, *C.C.* 11.
1885 ROBSON, A. W. MAYO, 8, Park Crescent, W., (C. 1893–6.) *Trans.* 15.
1897 ROGER-SMITH, HUGH, M.D., 1, College Terrace, Fitzjohn's Avenue, N.W. *Trans.* 1.
1889 ROLLESTON, HUMPHRY DAVY, M.A., M.D., 55, Upper Brook Street, Grosvenor Square, W. (C. 1899–1902.) *Trans.* 11. *C.C.* 9.
1897 ROSS, FREDERICK WILLIAM FORBES, M.D., 15, Gower Street, W.C.
1877 ROTH, BERNARD, 38, Harley Street, Cavendish Square, and "Wayside," Preston Park Avenue, Brighton. *Trans.* 1, *C.C.* 4.
1890 ROUGHTON, EDMUND WILKINSON, B.S., 38, Queen Anne Street, W. *Trans.* 1, *C.C.* 1.
1874 ROWLAND, EDWARD ROGER, Dordrecht, Wodehouse, S. Africa.
1904 ROWLANDS, R. P., M.S., 6, St. Thomas's Street, London Bridge, S.E. *C.C.* 2.

ELECTED

1898 RUSSELL, ALFRED ERNEST, M.D., B.S., 9, Wimpole Street, Cavendish Square, W. *C.C.* 1.

1900 RUSSELL, J. S. RISIEN, M.D., 44, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1903 RUSSELL, JAMES WILLIAM, M.D., 72, Newhall Street, Birmingham.

1887 RUTHERFOORD, H. T., M.B., Salisbury House, Taunton.

1885 RYLE, REGINALD JOHN, M.D., 15, German Place, Brighton.

1882 SAINSBURY, HARRINGTON, M.D., 52, Wimpole Street, W. (C. 1898–1901.)

1899 SANDIFER, HENRY STEPHEN, M.D., B.S., 11, Porchester Gardens, W.

1893 SANSOM, ARTHUR ERNEST, M.D., 84, Harley Street, W. *Trans.* 1, *C.C.* 1.

1873 SAVAGE, GEORGE HENRY, M.D., 3, Henrietta Street, W. (C. 1882–3.)

1886 SAVILL, THOMAS DIXON, M.D., 60, Upper Berkeley Street, W. *Trans.* 1, *C.C.* 6.

1899 SCHORSTEIN, GUSTAVE, M.B., 11, Portland Place, W.

1886 SCOTT, ALFRED, 15, German Place, Brighton.

1894 SCOTT, BERNARD, "Hartington," Poole Road, Bournemouth.

1892 SCOTT, RICHARD JAMES HERBERT, 28, Circus, Bath. *C.C.* 1.

1902 SEARS, ALFRED ERNEST, 33, Lee Terrace, Blackheath, S.E. *Trans.* 1.

1897 SEGUNDO, CHARLES SEMPILL DE, M.B., B.S., 6, Brook Street, Hanover Square, W.

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood Road, S.W.

1878 SEMON, Sir FELIX, C.V.O., M.D., 39, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.C.* 3.

1884 SHARKEY, SEYMOUR J., M.D., 22, Harley Street, W. (C. 1895–8.)

1898 SHAW, HAROLD BATTY, M.D., 7, Devonshire Street, Portland Place, W. *C.C.* 8.

1889 SHAW, LAURISTON ELGIE, M.D., 64, Harley Street, W. *C.C.* 1.

1875 SHERWOOD, ARTHUR PAUL, 8, Seaside Road, Eastbourne.

1903 SIMPSON, GRAHAM, The Sheffield Royal Hospital, Sheffield. *C.C.* 1.

1879 SKERRITT, EDWARD MARKHAM, M.D., Ivor House, Durdham Park, Clifton, Bristol. (C. 1895–8.) *Trans.* 2.

1872 SLIGHT, GEORGE, M.D., 14, Old Burlington Street, W.

1882 SMITH, E. NOBLE, 24, Queen Anne Street, W. *Trans.* 1, *C.C.* 2.

1896 SMITH, EBENEZER STANLEY, M.D., 68, Wimpole Street, Cavendish Square, W.

1888 SMITH, FREDERICK J., M.D., 138, Harley Street, W. (C. 1905– .) *Trans.* 2.

1884 SMITH, R. PERCY, M.D., 36, Queen Anne Street, Cavendish Square, W. (C. 1898–1901.)

1894 SMITH, THOMAS RUDOLPH, M.B., B.C., 25, Bridge Road, Stockton-on-Tees.

1893 SNAPE, ERNEST ALFRED, M.D., 41, Welbeck Street, W.

1868 SNOW, WILLIAM V., M.D., Richmond Gardens, Bournemouth.

ELECTED

1890 SOLLY, ERNEST, M.B., Strathlea, Harrogate, Yorks. *C.C.* 1.

1888 SPENCER, WALTER GEORGE, M.S., M.B. (*Hon. Secretary*), 35, Brook Street, W. (C. 1896-9. S. 1904– .) *Trans.* 13, *C.C.* 15.

1885 SPICER, FREDERICK, M.D., 17, Wimpole Street, Cavendish Square, W.

1888 SPICER, ROBERT HENRY SCANES, M.D., 28, Welbeck Street, W. *C.C.* 1.

1882 SPOONER, FREDERICK HENRY, M.D., 4, Maitland Place, Lower Clapton, N.E.

1896 SPURRELL, CHARLES, Medical Superintendent, Poplar and Stepney Sick Asylum, Devon's Road, Bromley-by-Bow, E.

1876 SQUIRE, A. BALMANNO, 24, Weymouth Street, W. *Trans.* 5, *C.C.* 4.

1892 STABB, EWEN CARTHEW, 57, Queen Anne Street, W. *C.C.* 1.

1879 STAPLES, FRANCIS PATRICK, Brigade-Surgeon, Army (retired). (Address uncommunicated.)

1896 STEWARD, FRANCIS JAMES, M.B., B.S., 133, Harley Street, W. *Trans.* 3, *C.C.* 1.

1874 **Stirling**, EDWARD C., M.D. [care of Messrs. ELDER & Co., 7, St. Helen's Place, E.C.], Adelaide, South Australia.

1899 STONE, WILLIAM GREAM, M.D., 93, Denmark Hill, S.E. *C.C.* 1.

1884 STONHAM, CHARLES, C.M.G., 4, Harley Street, W. *C.C.* 3.

1878 STRUGNELL, FREDERICK WILLIAM, Grove End House, Highgate Road, Highgate, N.W. (C. 1903– .) *C.C.* 1.

1878 **Sturge**, WILLIAM ALLEN, M.D., 29, Boulevard Dubouchage, Nice, France.

1894 SUTHERLAND, GEORGE A., M.D., 73, Wimpole Street, Cavendish Square, W. *Trans.* 1.

1882 SYMONDS, CHARTERS JAMES, M.S., 58, Portland Place, W. (C. 1888–9, 1901–4, S. 1898–1901.) *Trans.* 12, *C.C.* 23.

1876 SYMONDS, HORATIO PERCY, 35, Beaumont Street, Oxford.

1885 TAIT, EDWARD SABINE, M.D., 48, Highbury Park, N.

1885 TAIT, HENRY BREWER, Lincluden, Sunnyside Road, Hornsey Lane, N.

1891 TATE, WALTER WILLIAM HUNT, M.D., 32, Queen Anne Street, W.

1886 TAY, WARREN, 4, Finsbury Square, E.C.

1897 TAYLOR, E. CLAUDE, M.D., B.S., 181, Rosslyn Hill, Hampstead, N.W.

1875 **TAYLOR**, FREDERICK, M.D., 20, Wimpole Street, W. (S. 1879-81, C. 1882–4, V.P. 1894–7, T. 1901–3, P. 1903–5.) *Trans.* 17, *C.C.* 3.

1889 **Taylor**, HENRY HERBERT, 10, Brunswick Place, Hove, Sussex.

1890 TAYLOR, JAMES, M.D., 49, Welbeck Street, W. *C.C.* 3.

1882 TAYLOR, SEYMOUR, M.D., 16, Seymour Street, W. *Trans.* 1, *C.C.* 4.

1885 **Taylor**, W. C. EVERLEY, 34, Queen Street, Scarborough.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., 38, Cookridge Street, Leeds. (C. 1897–1900.)

1896 TEMPLETON, GEORGE, M.B., 10, Seymour Street, Portman Square, W.

1890 THANE, EDGAR HERBERT, M.D., Wagga-Wagga, New South Wales.

ELECTED

1886 THOMPSON, CHARLES HERBERT, M.D., 133, Harley Street, Cavendish Square, W.

O.M. THOMPSON, EDMUND SYMES, M.D., 33, Cavendish Square, W. (C. 1880–2.) *Trans.* 1.

1897 THOMSON, H. CAMPBELL, M.D., 34, Queen Anne Street, Cavendish Square, W. *C.C.* 1.

1894 THOMSON, STCLAIR, M.D., 28, Queen Anne Street, W. *Trans.* 2, *C.C.* 8.

1896 THORNE, WILLIAM BEZLY, M.D., 2, Harley Street, W. *C.C.* 2.

1872 THORNTON, WILLIAM PUGIN, 35, St. George's Road, Canterbury. *Trans.* 5.

1885 THURSFIELD, THOMAS WILLIAM, M.D., Selwood, Beauchamp Square, Leamington.

1899 TILLEY, HERBERT, M.D., 89, Harley Street, Cavendish Square, W. *C.C.* 2.

1891 TOMSON, W. BOLTON, M.D., Park Street West, Luton, Bedfordshire.

1897 TOOGOOD, F. SHERMAN, M.D., The Infirmary, 282, High Street, Lewisham, S.E.

1892 TOOTH, HOWARD HENRY, C.M.G., M.D., 34, Harley Street, W. *Trans.* 1.

1887 TOTSUKA, KANKAI, Tokio, Japan.

1897 TUBBY, ALFRED HERBERT, M.S., 25, Weymouth Street, Portland Place, W. *Trans.* 2, *C.C.* 5.

1900 TULLIS, GEORGE AUGUSTUS, M.D., Holmbush, Grove Road, Southsea.

1904 TURNER, GEORGE GREY, 31, Oxford Street, Newcastle-on-Tyne.

1882 TURNER, GEORGE ROBERTSON, 41, Half Moon Street, W. (C. 1897–1900.) *Trans.* 9. *C.C.* 6.

1904 TURNER, PHILIP, M.S., 8, St. Thomas's Street, London Bridge, S.E.

1888 TURNER, PHILIP DYMOCK, M.D., Sudbury, Ryde, Isle of Wight. *Trans.* 2.

1898 TURNER, WILLIAM, M.S., 17, Harley Street, Cavendish Square, W. *C.C.* 1.

1893 TURNEY, HORACE GEORGE, M.D., 68, Portland Place, W. *C.C.* 4.

1878 TYSON, WILLIAM JOSEPH, M.D., 10, Langhorne Gardens, Folkestone. (C. 1886–8, V.-P. 1900–2.) *Trans.* 8.

1881 UHTHOFF, JOHN CALDWELL, M.D., Wavertree House, Brunswick Place, Brighton. (C. 1898–1901.)

1868 VENNING, EDGCOMBE, 30, Cadogan Place, S.W. (C. 1876–8.) *Trans.* 2.

1890 VOELCKER, ARTHUR FRANCIS, M.D., B.S., 101, Harley Street, W. (C. 1900–3.) *Trans.* 2, *C.C.* 2.

1868 WAGSTAFFE, WILLIAM WARWICK, Purleigh, St. John's Hill, Sevenoaks. (C. 1878.)

1886 **WAINEWRIGHT**, BENJAMIN, M.B., C.M. (address uncommunicated).

1885 WAKLEY, THOMAS, jun., 16, Hyde Park Gate, S.W.

1899 WALKER, HENRY ROE, 8, Harley Street, W.

1896 WALLACE, CUTHBERT SYDNEY, M.B., 26, Upper Wimpole Street, W. (C. 1902–5.) *Trans.* 7, *C.C.* 5.

1890 WALLIS, FREDERICK CHARLES, M.B., B.C., 107, Harley Street, W. (C. 1902–5.) *Trans.* 5, *C.C.* 10.

ELECTED

1888 WALTERS, FREDERICK RUFENACHT, M.D., Crooksbury Sanatorium, Farnham, Surrey. *C.C.* 2.

1896 WARD, EDWARD, M.B., 22, Park Place, Leeds. *Trans.* 1.

1904 WARING, HOLBURT J., M.S., 37, Wimpole Street, Cavendish Square, W.

1888 Warner, PERCY, Rydal, Woodford Green, Essex.

1891 WATERHOUSE, HERBERT FURNIVALL, M.D., C.M., 81, Wimpole Street, W. (C. 1905– .)

1902 WATSON, CHARLES GORDON, 44, Welbeck Street, Cavendish Square, W. *C.C.* 1.

1896 WEBER, FRED. PARKES, M.D., 19, Harley Street, W. *Trans.* 2, *C.C.* 11.

O.M. WEBER, Sir HERMANN D., M.D., 10, Grosvenor Street, W. (C. 1867–71, V.P. 1873–5.) *Trans.* 9.

1882 WEST, SAMUEL, M.D., 15, Wimpole Street, W. (C. 1887–9.) *Trans.* 16, *C.C.* 5.

1895 WETHERED, FRANK J., M.D., 83, Harley Street, W.

1874 WHEELHOUSE, CLAUDIUS GALEN, Cliffe Point, Filey, Yorks. *Trans.* 1.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 22, Cadogan Gardens, S.W.

1882 WHITE, EDWIN FRANCIS, Westlands, 280, Upper Richmond Road, Putney, S.W.

1890 WHITE, GILBERT B. MOWER, M.B., B.S., 112, Harley Street, W.

1883 WHITE, WILLIAM HALE, M.D. 65, Harley Street, W. (C. 1887–9, 1896–9, S. June, 1893–6.) *Trans.* 17, *C.C.* 4.

1883 WHITE, WILLIAM HENRY, M.D., 43, Weymouth Street, W. *Trans.* 1. *C.C.* 1.

1894 WHITELOCKE, RICHARD HENRY ANGLIN, M.B., C.M., 6, Banbury Road, Oxford.

1897 WHITFIELD, ARTHUR, M.D., 21, Bentinck Street, Manchester Square, W.

1882 WHITTLE, EDWARD GEORGE, M.D., 9, Regency Square, Brighton.

1871 WIGHT, GEORGE, M.B., C.M., 428, Liverpool Road, N.

1879 WILCOX, HENRY, M.B., Newlyn, Fleet, Hants.

1894 WILKIN, GRIFFITH CHARLES, Conway House, Paignton, South Devon.

1884 WILLCOCKS, FREDERICK, M.D., 14, Mandeville Place, W. *C.C.* 1.

O.M. WILLETT, ALFRED, 36, Wimpole Street, W. (C. 1872–5, V.P. 1889–91.) *C.C.* 2.

1888 WILLIAMS, CAMPBELL, 18, Queen Anne Street, Cavendish Square, W. *Trans.* 2.

O.M. WILLIAMS, CHARLES THEODORE, M.D., 2, Upper Brook Street, W. (C. 1877–9, V.P. 1889–90.) *Trans.* 8.

1888 WILLIAMS, DAWSON, M.D., 2, Agar Street, Strand, W.C. (C. 1893–7.)

1903 WILLIAMS, LEONARD, M.D., 8, York Street, Portman Square, W. *C.C.* 1.

1890 WILLIAMS, W. ROGER, Beaufort House, Clifton Down, Clifton, Bristol.

O.M. WILLIS, FRANCIS, M.D., The Spa, Braceborough, Stamford.

1893 WILLS, JOSEPH PEARCE BUDGETT, M.D., Chintings, Bexhill-on-Sea.

ELECTED

1886 WILSON, ALBERT, M.D., Minto House, South Woodford, Essex. *Trans.* 1, *C.C.* 2.

1888 Wilson, CLAUDE, M.D., C.M., Belmont, Tunbridge Wells. (C. 1905– .) *Trans.* 2.

1891 WILSON, THEODORE STACEY, M.D., C.M., 27, Wheeley's Road, Edgbaston, Birmingham. *Trans.* 2.

1890 WOOD, NEVILLE, 42, Elvaston Place, Queen's Gate, S.W.

1883 WOODCOCK, JOHN ROSTRON, Darlington Court, North Road, Bath.

1879 WOODWARD, GEORGE P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1894 WOOLLETT, CHARLES JEROME, "Ambleside," Streatham, S.W.

1884 WORTS, EDWIN, 6, Trinity Street, Colchester.

1892 WYNTER, WALTER ESSEX, M.D., B.S., 27, Wimpole Street, Cavendish Square, W. *C.C.* 4.

1872 YEO, I. BURNEY, M.D., 44, Hertford Street, W. (C. 1881–3.) *Trans.* 8.

[*It is requested that any change of Title or Residence be communicated to the Secretaries before the* 1*st of July in each year, in order that the list may be made as correct as possible.*]

LIST OF MEMBERS.

ORIGINAL MEMBERS (ALPHABETICALLY).

Henry Arnott.
Richard Barwell.
Henry Charlton Bastian, M.D., F.R.S.
Sir Wm. Henry Broadbent, Bart., K.C.V.O., M.D., F.R.S.
Thomas Bryant.
Thomas Buzzard, M.D.
Sir William Selby Church, Bart., K.C.B., M.D.
Edward Clapton, M.D.
John Couper.
William Howship Dickinson, M.D.
Sir Dyce Duckworth, M.D.
Timothy Holmes.
Jonathan Hutchinson, F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
John Langton.
Arthur Trehern Norton, C.B.
Frederick William Pavy, M.D., F.R.S.
Sir Richard Douglas Powell, Bart., M.D., K.C.V.O.
Sydney Ringer, M.D., F.R.S.
Sir Thomas Smith, Bart., K.C.V.O.
Edmund Symes Thompson, M.D.
Sir Hermann D. Weber, M.D.
Alfred Willett.
Charles Theodore Williams, M.D.
Francis Willis, M.D.

ARRANGED ACCORDING TO DATE OF ELECTION.

1868 Sir Constantine Holman, M.D.
Christian G. H. Bäumler, M.D.
James Grey Glover, M.D.
T. Henry Green, M.D.
Howard Marsh, M.C.
Charles Prentis.
Edgcombe Venning.
John Ford Anderson, M.D.

1868 George Granville Bantock, M.D.
George Charles Bright, M.D.
Frank W. Cooper.
Edward Hooper May, M.D.
William Warwick Wagstaffe.
James Edward Pollock, M.D.
Franz Oppert, M.D.
William V. Snow, M.D.

1868 Charles Drage, M.D.
Frederick Royston Fairbank, M.D.
Michael W. Rice, M.D.
1869 J. Warrington Haward.
Henry Frederick Augustus Goodridge, M.D.
Olliver Thomas Duke, M.B.
1871 Alfred Playne, M.B.
George Wight, M.B.
Ebenezer Diver, M.D.
1872 I. Burney Yeo, M.D.
Henry Harris, M.D.
William Pugin Thornton.
Sir G. Anderson Critchett.
J. C. J. Fenwick, M.D.
Andrew S. Myrtle, M.D.
Sir William Bartlett Dalby, M.B.
Thomas Ridge Jones, M.D.
George Slight, M.D.
1873 William Julius Mickle, M.D.
David Lloyd Roberts, M.D.
George Henry Savage, M.D.
Edwin Chisholm, M.D.
Thomas Churton, M.D.
1874 John Hammond Morgan, C.V.O.
Edward R. Rowland.
Claudius Galen Wheelhouse.
Edward C. Stirling, M.D.
Sir William Henry Bennett, K.C.V.O.
William Brown Holderness.
Andrew Clark.
1875 Sir Thomas Barlow, Bart., K.C.V.O., M.D.
Sidney Coupland, M.D.
Clinton T. Dent.
C. D. B. Hale.
Edward Liveing, M.D.
Rickman John Godlee, M.S.
Arthur Paul Sherwood.
James Frederic Goodhart, M.D.
Sir William Richard Gowers, M.D., F.R.S.
William Smith Greenfield, M.D.
Sir Shirley F. Murphy.
Herbert W. Page.
Frederick Taylor, M.D.
1876 Arthur E. J. Barker.
Horatio Percy Symonds.
A. Balmanno Squire.
David White Finlay, M.D.
Furneaux Jordan.
R. Clement Lucas, B.S.
George Buckston Browne.
1876 Arthur Edwin Temple Longhurst, M.D.
1877 Robert Hogarth Clay, M.D.
A. Pearce Gould, M.S.
Henry Radcliffe-Crocker, M.D.
David B. Lees, M.D.
William Ewart, M.D.
Henry Morris, M.B.
Henry Ambrose Lediard, M.D.
Bernard Roth.
Henry Hugh Clutton, M.C.
Malcolm Alex. Morris.
1878 George P. Field.
Thomas Warner Lacey.
Thomas Colcott Fox, M.B.
Sir Felix Semon, C.V.O., M.D.
Henry de Fonmartin, M.D.
C. H. Golding-Bird, M.B.
Donald William Charles Hood, M.D.
Lord Lister, P.C., O.M., F.R.S.
F. de Havilland Hall, M.D.
William Allen Sturge, M.D.
William Joseph Tyson, M.D.
Charles Robert Bell Keetley.
William Appleton Meredith, C.M.
Robert Bridges, M.B., M.A.
Frederick William Strugnell.
1879 William Edward Burton.
James Magill, M.D.
Edward Markham Skerritt, M.D.
Henry Wilcox, M.B.
Sir Stephen Mackenzie, M.D.
William Harrison Cripps.
Francis Patrick Staples.
Geo. Courtenay Henderson, M.D.
Henry Davy.
Thos. Walter Harropp Garstang.
Charles W. Mansell Moullin.
John Reuben Lunn.
George P. M. Woodward, M.D.
Robert Peel.
Frederic S. Dennis, M.D.
1880 T. Mark Hovell.
Wyndham Cottle, M.D.
Henry Francis Baker.
Bernard O'Connor, M.D.
Charles Edward Beevor, M.D.
1881 George Henry Makins, C.B.
Robert William Burnet, M.D.
James Kingston Fowler, M.D.
Charles Edward Harrison, M.B.
Malcolm Macdonald McHardy.

1881 Rushton Parker.
Montagu Lubbock, M.D.
William Pasteur, M.D.
John Caldwell Uhthoff, M.D.
Henry Treutham Butlin.
H. A. Powell, M.A.
1882 George Robertson Turner.
E. Noble Smith.
Robert William Goldie.
Frederick Charles Barker, M.D.
Daniel Colquhoun, M.D.
Seymour Taylor, M.D.
Philip Henry Bindley, M.B.
Edward George Whittle, M.D.
D. H. Goodsall.
Frederick Henry Spooner, M.D.
Frederick Haycraft Berry, M.D.
Herbert Collier. M.D.
Samuel West, M.D.
Emile Emond, M.D.
Charters James Symonds, M.S.
C. F. Coxwell, M.B.
Augustus Joseph Pepper. M.B.
Harrington Sainsbury, M.D.
Edwin Francis White.
1883 Anthony A. Bowlby, C.M.G.
Percy Kidd, M.D.
William Henry White, M.D.
Hubert Montague Murray, M.D.
Robert Fitzroy Benham.
William Henry Allchin, M.D.
John Mitchell Bruce, M.D.
William Arbuthnot Lane, M.S.
Bernard Pitts.
William Hale White, M.D.
William Coode Adams, M.B.
Robert Leamon Bowles, M.D.
George Henry Jackson.
Thomas Laurence Read.
Frederick Thomas Roberts, M.D.
Charles Alfred Ballance, M.B., M.S.
John Rostron Woodcock.
1884 Frederick Willcocks, M.D.
R. Percy Smith, M.D.
Edgar Duke.
John Mackern, M.B.
Paul M. Chapman, M.D.
Wilmot Parker Herringham, M.D.
Philip Henry Pye-Smith, M.D., F.R.S.
Charles Stonham, C.M.G.
Dudley Wilmot Buxton, M.D.

1884 Edwin Worts.
Seymour J. Sharkey, M.D.
John James Pringle, M.B.
Frederick Lucas Benham, M.D.
Walter Edmunds, M.C.
Stephen Paget.
Sir Lambert Hepenstal Ormsby, M.D.
Edwin Leonard Adeney, M.D.
Sir Victor Horsley, F.R.S.
Henry Carr Maudsley, M.D.
Bilton Pollard, B.S.
1885 Frederick Spicer, M.B.
Herbert Larder.
James Berry, B.S.
Fred. Walker Mott, M.D., F.R.S.
George Newton Pitt, M.D.
W. C. Everley Taylor.
Sidney Phillips, M.D.
A. W. Mayo Robson.
Thomas Wakley, jun.
Herbert William Allingham.
Thomas William Thursfield, M.D.
Alexander Dalton Murray, M.B.
Robert Maguire, M.D.
Robert Alexander Gibbons, M.D.
Henry Brewer Tait.
Richard Caton, M.D.
Arthur Henry Robinson, M.D.
Edward Sabine Tait, M.B.
William Bruce Clarke, M.B.
Charles Barrett Lockwood.
Reginald J. Ryle, M.D.
J. Michell Clarke, M.D.
Henry George Armstrong.
Roderick Maclaren, M.D.
W. Watson Cheyne, C.B., F.R.S.
1886 Thomas Dixon Savill, M.D.
Benjamin Wainewright, M.B.
Waren Tay.
William Henry Battle.
James Hardie, M.D.
Francis Henry Hawkins, M.D.
R. Hingston Fox, M.D.
John Ward Cousins, M.D.
T. Pridgin Teale, F.R.S.
H. Lankester, M.D.
Arthur T. Davies, M.D.
Charles Herbert Thompson, M.D.
Henry Handford, M.D.
Alfred Scott.
Albert Wilson, M.D.
1887 Archibald E. Garrod, M.D.
H. T. Rutherfoord, M.B.

1887 Kankai Totsuka.
Thomas Oliver, M.D.
Francis George Penrose, M.D.
Samuel Herbert Habershon, M.D.
Oswald Auchinleck Browne, M.D.
Joseph Arderne Ormerod, M.D.
J. H. E. Brock, M.D., B.S.
Francis William Clark.
A. H. Weiss Clemow, M.D., C.M.
E. Hurry Fenwick.
R. Lawford Knaggs, B.C.
John D. Malcolm, M.B., C.M.
Sidney Martin, M.D., B.S., F.R.S.
Thomas Horrocks Openshaw, C.M.G., M.B.

1888 A. G. Barrs, M.D.
J. W. Batterham, M.B., B.S.
Montagu Handfield-Jones, M.D.
Alfred Rice Oxley, M.D.
Robert Henry Scanes Spicer, M.D.
Campbell Williams.
Frederic S. Eve.
Alexander Morison, M.D.
Frederick Page, M.D.
Frederick J. Smith, M.D.
Frederick R. Walters, M.D.
Claude Wilson, M.D., C.M.
Charles H. Gage-Brown, M.D.
J. H. Menzies.
Frank Ernest Roberts.
Robert Ashton Bostock.
Hugh Armstrong.
Hyde Marriott, M.B.
Percy Warner.
J. T. James, M.D.
Edwin A. Barton.
W. Page May, M.D.
Philip D. Turner, M.D.
Dawson Williams, M.D.
Augustus W. Addinsell, M.B., C.M.
John Anderson, C.I.E., M.D.
Henry French Banham, M.D.
David James Mason, M.D., C.M.
Walter G. Spencer, M.B., M.S.

1889 Theodore Dyke Acland, M.D.
Raymond Johnson, M.B., B.S.
H. D. Rolleston, M.A., M.D.
P. MacBride, M.D.
D. Newman, M.D.
Herbert Elwin Harris, M.B.
John E. Ranking, M.D.
Edward Ashby Fardon.
Stanley Boyd, B.S.

1889 George Ezra Halstead, M.D., B.S.
Henry Herbert Taylor.
John Duncan, M.D.
Wm. Wallis Ord, M.D., B.Ch.
Leonard Arthur Bidwell.
Arthur J. M. Bentley, M.D.
Francis R. B. Bisshopp, M.D., B.Sc.
Henry Percy Dean, M.B.
Louis Albert Dunn, M.S.
Herbert Pennell Hawkins, M.D., B.Ch.
Lauriston Elgie Shaw, M.D.

1890 John Rose Bradford, M.D., F.R.S.
J. Fletcher Little, M.B.
Robert Robertson, M.D.
Ernest Solly, M.B.
James Taylor, M.D.
Francis O. Buckland, B.A., M.B., C.M.
Edmund W. Roughton, B.S.
James Calvert, M.D.
H. Roxburgh Fuller, M.D.
Arthur F. Voelcker, M.D.
Neville Wood.
W. Roger Williams.
Gilbert B. M. White, M.B., B.S.
Frederick Charles Wallis, M.B., B.C.
Alfred Parkin, M.S.
George A. Hawkins-Ambler.
James Harper, M.D.
Walter Henry Brown.
John Walter Carr, M.D.
Ernest Le Cronier Lancaster, M.B., B.Ch.
Sir Patrick Manson, K.C.M.G., M.D., F.R.S.
Miles Miley, M.A., M.B.
Edgar Herbert Thane, M.B.
Charles William Chapman, M.D.
Michael G. Foster, M.A., M.D.

1891 Frederic François Burghard, M.D., M.S.
Roger Neville Goodman, M.B.
Herbert Furnivall Waterhouse, M.D., C.M.
Walter William Hunt Tate, M.D.
Greville MacDonald, M.D.
J. Kingston Barton.
J. Dundas Grant, M.D.

1891 W. Kington Fyffe, M.B., B.C.
Albert Carless, M.B., M.S.
W. Bolton Tomson, M.D.
Harry Littlewood, M.D.
Hector W. G. Mackenzie, M.A., M.D.
Chas. Percival White, M.B., B.C.
Arnold Caddy.
Theodore Stacey Wilson, M.D., C.M.

1892 William Hunter, M.D.
Frank Thomas Paul.
Walter Essex Wynter, M.D., B.S.
Damer Harrison.
John Alfred Masters, M.D.
Walter Gifford Nash.
John Stephenson Selwyn-Harvey, M.D.
Ewen Carthew Stabb.
Edward John Lewis, M.B., B.C.
Henry Betham Robinson, M.D. M.S.
Richard James Herbert Scott.
Howard Henry Tooth, C.M.G., M.D.

1893 John Ernest Paul, M.B.
James William Bond, M.D.
Harry Campbell, M.D.
W. Soltau Fenwick, M.D.
Ernest Alfred Snape, M.D.
Lewis G. Glover, M.B., B.C.
William Bracewell Mercer, M.B., B.C.
Robert Henry Cole, M.D.
Donald Rose Paterson, M.D., C.M.
Walter Spencer Anderson Griffith, M.D.
Alfred Theodore Rake, M.B., B.S.
James William Browne, M.B.
George Redmayne Murray, M.D., M.A.
R. J. Bliss Howard, M.D.
Henry Jones Curtis, M.D., B.S.
Henry Albert Caley, M.D.
John Henry Dauber, M.B.
Theodore Henry Ionides, M.B., B.S.
Arthur Foxwell, M.D.
Horace George Turney, M.D.
Henry Grabham Lys, M.D.
Arthur Ernest Sansom, M.D.
John Henry Bryant, M.D.

1893 G. F. Johnston, M.D.
Willmott H. Evans, M.D., B.S.
Charles Gibbs.
Robert Stephen Charsley.
Joseph Pearce Budgett Wills, M.D.
Arnold Lawson, M.D.

1894 Herbert Morley Fletcher, M.D.
Edward Wilberforce Goodall, M.D.
Harry Gilbert Barling.
Ernst Michels, M.D.
Charles Jerome Woollett.
StClair Thomson, M.D.
George A. Sutherland, M.D.
John Murray.
Richard Henry Anglin Whitelocke, M.B., C.M.
John Attlee, M.D., B.C.
Griffith Charles Wilkin.
Thomas Hugh Dickson, M.B., B.C.
C. Gordon Brodie.
Bernard Scott.
John Porter Parkinson, M.D.
Thomas Rudolph Smith, M.B., B.C.
Arthur Pearson Luff, M.D.

1895 Herbert Murray Ramsay.
Arthur G. Phear, M.D., B.C.
Ernest E. Lewis, M.D.
Karl Grube, M.D.
Gerald R. Baldwin.
James Galloway, M.D.
F. B. Willmer Phillips, M.B.
Charles Henry Hough.
Leonard George Guthrie, M.D.
James Ernest Lane.
Frank J. Wethered, M.D.
Richard Ackerley.

1896 Charles Spurrell.
Cuthbert Sidney Wallace, M.B.
Charles R. Box, M.D.
Walter Stacy Colman, M.D.
Edward Ward, M.B.
Norman Dalton, M.D.
Fred. Parkes Weber, M.D.
Francis James Steward, M.B., B.S.
William Bezly Thorne, M.D.
Douglas Drew, M.D., B.S.
Surg.-Capt. W. R. Crooke-Lawless, A.M.S., M.D.
William Elliot Risdon, M.D.
Priestley Leech, M.D.
Ebenezer Stanley Smith, M.D.

1896 Edward Cooper Bentham.
Robert Herbert Mills-Roberts, C.M.G.
Jonathan Hutchinson, jun.
Frederick E. Batten, M.D.
Rutherford John Pye-Smith.
George Templeton.
Bertram Louis Abrahams, M.B., B.Sc.

1897 Raymond H. P. Crawfurd, M.D.
John Henry Deane.
Percy Furnivall.
Arthur Whitfield, M.D.
Frederick William Forbes Ross, M.D.
Harold Low, M.B.
Charles Ralph Keyser.
George Bertram Hunt, M.B.
Hugh Roger-Smith, M.D.
Alfred Herbert Tubby, M.S.
Charles Lamplough.
H. Campbell Thomson, M.D.
Alfred P. Hillier, M.D., C.M.
F. Sherman Toogood, M.D.
E. Claude Taylor, M.D.
Charles Humphrys.
Charles Sempill de Segundo, M.B.
Sir Alfred Downing Fripp, C.B., M.V.O., M.S.
Chichester Gould May, M.D.

1898 J. J. Perkins, M.D.
James Jackson Clarke, M.B.
Ernest Herbert Cobb.
Sydney Backhouse Hulke.
Thomas Herbert Kellock.
William Darley-Hartley.
Robert Jones.
Arthur Philip Beddard, M.D.
Alfred Ernest Russell, M.D., B.S.
William Cecil Bosanquet, M.D.
Jonathan F. C. H. Macready.
Arthur A. H. Partridge, M.B., M.Ch.
Leonard Avery.
Herbert Challice Crouch.
William Turner, M.B., B.S.
Harold Batty Shaw, M.D.
Joseph Stewart Richards, M.D., B.S.
William Henry Breffit Brook, M.D., B.S.

1899 George Edward Rennie, M.D.
V. Warren Low, M.D., B.S.
Rayner Derry Batten, M.D., B.S.
Charles E. Forbes Mouat-Biggs.
Rutherford Morison.
R. Murray Leslie, M.D.
Herbert Tilley, M.D.
Charles Owen Fowler, M.D.
Cyril Ogle, M.B.
Charles Herbert Fagge, M.S.
Claude Somerville Hawkes.
Henry Stephen Sandifer, M.D., B.S.
Marcus Marwood Bowlan, M.B., B.Sc.
Alfred Eïd, M.D.
Karl Fürth, M.D.
Wm. Gream Stone, M.D.
Andrew Duncan, M.D.
Edmond Lucien De Chazal, M.D.
Henry Roe Walker.
Gustave Schorstein, M.B.
Edward Collingwood Andrews, M.D.

1900 Berkeley George Andrew Moynihan, M.S.
Harry Cooper, M.D.
George Augustus Tullis, M.D.
Griffith Lloyd Roberts, M.B., C.M.
Edward Percy Paton, M.S.
George Steele Perkins, M.D.
John Arthur Hayward, M.D.
Wilfred J. Hadley, M.D.
John Fawcett, M.D.
Arthur Bousfield, M.B.
J. S. Risien Russell, M.D.
Cecil H. Leaf, M.B.
Josef Paul Zum Busch, M.D.

1901 Ernest George Leopold Goffe, M.D.
Arthur Hamilton Nicholson Lewers, M.D.
Charles Reissmann, M.D.
Adrian Caddy, M.B.
Herbert William Carson.
Thomas Henry Morse.
Laurie Hugh McGavin.
F. U. Menzies.
Thomas Rupert Hampden Bucknall, M.S.
Edward Angas Johnson, M.B.

List of Members arranged according to Date of Election.

1901 Percival Horton-Smith-Hartley, M.D.
Sir Hugh Reeve Beevor, Bart., M.D.
Theo. N. Kelynack, M.D.
Thomas Carwardine, M.S.
Bowie Campbell Gowan.
John J. Douglas, M.D.
Lieut.-Col. Alfred Keogh, C.B., M.D.
Harold Shuttleworth Barwell.

1902 Charles Henry Fennell, M.D.
Harold L. Barnard.
Thomas Percy Legg, M.B.
Andrew Elliot, M.D.
Sir James Reid, Bart., G.C.V.O., K.C.B.
F. W. Price, M.B.
Alfred Ernest Sears.
Reginald F. Jowers.
Cecil Marriott, M.B.
Edward Farquhar Buzzard, M.D.
Donald John Armour, M.B.
Edred Moss Corner, B.Sc., M.B.
Herbert French, M.D.
S. Vere Pearson, M.B., B.C.
Lawrence Jones, M.B.
Thomas Crisp English.
Walter Fedde Fedden, M.B.
David Nabarro, M.D.
Charles A. Morton.
Victor Evelyn Collins, M.B.
Leonard S. Dudgeon.
R. O. Moon, M.D.
Charles Gordon Watson.
Charles Firmin Cuthbert.

1903 Raoul Ernest Delbruck, M.B.
Clive Riviere, M.D.
Edward A. Gates, M.D.
E. Graham Little, M.D.
Percy Robert Cooper, M.D., B.Sc.
Leonard Williams, M.D.
Ernest Lloyd Jones, M.D.
John Basil Hall, M.C.
Thomas Reuell Atkinson, M.D.
Graham Simpson.
Edgar Beaumont.
James William Russell, M.D.
Philip Maynard Heath, M.B.
Wilfred Harris, M.D.
Arthur S. Morley.
Hugh Rigby, M.S.

1904 Holburt J. Waring, M.S.
Charles Bolton, M.D.
R. P. Rowlands, M.S.
H. A. Ballance, M.S.
Frederic W. Longhurst.
H. S. D. Browne, M.B., B.C.
Philip Turner, M.S.
George Herbert Graham, M.D.
Holland John Cotton, M.D., C.M.
George Grey Turner, M.B., M.S.
John English Harburn.
Oswald Lacy Addison, M.B.
A. Ernest Jones, M.D., B.Sc.

1905 Harold A. T. Fairbank, M.S.
David Forsyth, M.D.
E. Rock Carling, M.B., B.S.
Joseph Cunning, M.B., B.S.
William Henry Bailey, M.D., D.P.H.

REPORT

OF THE

COUNCIL OF THE CLINICAL SOCIETY,

MAY, 1905.

THE Council is glad to report the continued prosperity of the Society. Numerous papers of considerable clinical interest have been presented, and the attendances, on clinical evenings more especially, have been well maintained.

The death-roll of the Society this year is a long and an unusually sad one, including as it does the names of several well known members of the Society and of the medical profession, who have been taken away in the prime of life. The names are—Sir John Simon, Mr. Herbert W. Allingham, Dr. W. Lee Dickinson, Dr. Julian Evans, Mr. D. M. Forbes, Dr. T. Gilbart-Smith, Dr. Charles Kelly, Dr. Angel Money, Mr. W. J. Penny, and Mr. A. Quarry Silcock.

Ten ordinary members have been admitted and there have been eleven resignations. There are at present 19 Honorary and 589 Ordinary Members.

The Treasurer's Statement shows the financial position of the Society to be satisfactory.

THE CLINICAL SOCIETY OF LONDON.

Statement of Cash Receipts and Payments from the 1st May, 1904, to the 30th April, 1905.

G. H. MAKINS, Esq., C.B., *Treasurer.*

RECEIPTS.	£	s.	d.
Balance, 1st May, 1904	138	16	2
182 Subscriptions at £1 1s. received by Collector — £191 2 0			
172 Subscriptions at £1 1s. received by Bank — 180 12 0			
354 —	371	14	0
22 Subscriptions at 10s. 6d. received by Collector — 11 11 0			
1 Subscription at 10s. 6d. received by Bank — 0 10 6			
	12	1	6
6 Admission Fees at £2 2s.	12	12	0
6 „ „ at £1 1s.	6	6	0
4 Composition Fees	21	0	0
Sale of *Transactions*	12	13	0
Dividend on Consols (£640)	15	4	0
	£590	6	8

PAYMENTS.	£	s.	d.	£	s.	d.
Cost of *Transactions*, Vol. XXXVII:						
Printing, paper, binding, delivery, &c.	£135	2	4			
Illustrations	38	18	0			
				174	0	4
Meetings:						
Rent of Rooms	105	0	0			
Hire of Microscopes	2	10	0			
Hire of Epidiascope	5	4	0			
A. Tapson (attendance)	7	0	0			
Refreshments	33	8	0			
				153	2	0
Cost of purchase of Consols (£28 18s. 2d.)				26	5	0
Treasurer and Hon. Secretaries:						
Secretarial assistance	26	5	0			
Commission to Collector	15	15	0			
				42	0	0
Printing and Stationery				79	4	7
Purchase of Cyclostyle				3	0	0
Petty Cash Expenditure				10	11	7
Balances:						
At Bank	100	14	11			
Petty Cash	1	8	3			
				102	3	2
				£590	6	8

FREDERICK TAYLOR, *President.*
G. H. MAKINS, *Treasurer.*
W. PASTEUR, *Hon. Secretary.*

Examined with the vouchers and found correct,
HY. BETHAM ROBINSON,
W. ESSEX WYNTER, } *Auditors.*
May 9th, 1905.

PRESIDENTIAL ADDRESS

DELIVERED AT THE

CLINICAL SOCIETY OF LONDON

ON OCTOBER 14TH, 1904

BY

FREDERICK TAYLOR, M.D.,

PRESIDENT.

GENTLEMEN,—It has been customary for the President of the Society at least once in his presidential career to address the members on some matters of interest either in regard to the work of the Society, or to the profession of medicine, or to the study of medical science. In conforming to this excellent rule, on the present occasion, I must first thank you for the honour you have done me in electing me a second time as President. I cannot pretend that the work of the President is arduous, but there are duties to perform, and I hope I may take my re-election as an indication that the duties have been performed at least to your satisfaction.

Occupying the Chair, as I think I have done regularly at every meeting through the Session, I have been led to observe many points in the working of the Society, and to think over them with regard to their bearing on the welfare and progress of the Society.

The welfare and success of the Society may be estimated in different ways—for instance by the number of its members, by the frequency of attendance of members at the meetings, and by the number and character of the papers read, or the amount and excellence of other material brought before the meetings.

The numbers of members show a satisfactory increase. In December, 1884, according to the Council's report, there were 382 members; in May, 1894, there were 535 members; and in May, 1903, there were 608, an increase of 60 per cent. in nineteen years. During this last period of ten years there has been an almost uniform addition of a few members each year, the balance of course over losses by retirement and decease. But in the last session there has been a slight fall, the numbers in May last being only 599.

During the last ten years the attendances have maintained a nearly uniform rate—that is, the variations above and below the average have been quite small. The average attendance for the last ten years has been 50·5 persons on each evening, of whom 7½ are visitors. In calculating this average I have not included an Extraordinary Meeting which took place on March 30, 1896, when Professor Sylvanus Thompson demonstrated and explained the Röntgen rays from the physical side, to a meeting of 200, of whom 112 were members and 88 were visitors.

The attendances of the last two years have been slightly below the average—those of the two preceding years were above the average,—and in the year 1899—1900 the attendances were very much below. It may be recalled that this was the first year of the South African War, when many surgeons and medical men were out of England, and this fact very likely had some influence, both direct and indirect, in this result.

It is well-known that the most popular evenings of the Clinical Society are those at which the clinical cases are exhibited. For some years now four evenings have been set apart for these exhibitions, and an analysis of the attendances on those evenings during the last nine years show how much above the average they are. Eighty, 90, or 100 members and visitors have frequently come on these occasions, and the average of all these thirty-six attendances is:

78 against 50 for the average of all the meetings, of which
13 is the average of the visitors against 7½, and
65 is the average of attendance of members against nearly 43.

Of these exhibition nights I shall have to speak later.

But it is gratifying to see that in spite of the increasing number of Medical Societies, whether determined by the specialisation of our work, as in the case of the Laryngological and Dermatological Societies, or by local professional enthu-

siasm, as in the case of Medical Societies established in the less central parts of London, the attendances at this Society have not materially lessened in recent times.

Another point of interest was the number of papers read during the Session, and without going into any detail it is sufficient to say that since the establishment of the four clinical evenings, the number of papers has ranged from twenty-eight to thirty-seven, which represents two, three, or four papers at each meeting. But it is, of course, obvious that the supply of papers cannot be increased indefinitely. The charm of a paper is often its power of eliciting a discussion, and a good discussion postpones the papers which are to follow; so that a small number of papers may be the result of good discussions, and conversely a large number of papers may mean that the debates have been meagre and badly sustained.

The next subject to which I have directed my attention,—indeed, it was the first in point of time which occurred to me to examine,—is the nature of the communications made to the Society. And first of all I naturally drew the broad distinction between medical and surgical communications, not in any spirit of emulation or jealousy, but from the feeling that surgical communications more often represented advances in manipulation and operative treatment, whereas medical papers were more often the means of bringing before the profession observations of diagnostic importance, or researches in clinical pathology.

So far as number is concerned I expected to find, from the recent great strides which surgery has made in directions scarcely approached twenty years ago, that the surgical papers were in excess of the medical, and this is indeed true, for during ten years, from 1893 to 1903, the papers which can be classed as medical are 151, those as surgical 194—that is, they are very nearly in the proportion of three medical to four surgical. What underlay my curiosity with regard to to this point was the desire to know to what extent the Clinical Society was directly contributing to the knowledge of the treatment of disease, and to ascertain this point a closer analysis of the subjects of both medical and surgical papers is required; and yet not a very close analysis, for it very soon appears that only about one-fourth of the cases classed as medical are brought forward on account of the treatment, whereas only one-fourth of the surgical cases are not brought forward for that reason.

The late Mr. Hulke, in a former Presidential address in October, 1893, made a similar observation, saying, "If I may venture to express an opinion in regard to progress made by medicine, in the presence of so many better instructed than myself, it is that a greater advance has been made in the direction of exact diagnosis than in the way of treatment," and he goes on to illustrate this by a reference to recent researches in the domain of nervous diseases.

To the credit of medicine it may be observed, and, of course, is well known, that a large proportion of the material for the cases of surgical operation has been provided by the physicians.

I do not wish it to be understood that the question of treatment is never mentioned in the papers which I have classed as not being therapeutical, but only that in them the clinical or pathological features of the case or cases have obviously been the main reason for reading the paper before the Society.

Whatever praise may be due to surgeons for the wonderful advances they have made since Lord Lister's early work, and whatever credit physicians may receive for their judgment in calling in at the right time the manipulative and operative assistance which they deny to themselves the privilege of rendering, it must surely be admitted that surgery is not an ideal method of treatment for the majority of complaints, and that we should all of us avoid it absolutely if we could only find that other less disagreeable means would bring about an equally satisfactory result. The collection and administration of the Cancer Research Fund is evidence that the want of something more than the power to remove cancerous tumours at the right time is widely and seriously felt. What is aimed at is prevention, for the medical cure of cancer has long been regarded as so hopeless and impossible that he who professes to have in his hands the means of such cure has been looked at with something more than disfavour by his medical brethren.

But there are plenty of ailments in dealing with which there is left a sufficient interval between prevention on the one hand, and the manipulations of surgery on the other, to allow of the methods of what is known as pure medicine or medical treatment.

I wish to inquire whether as much progress has been made in these methods as regards the direct treatment of disease

as has been made in surgery, or as we should like to see. Were I addressing the public it might be politic to lay before them the facts, which show that progress has really been made in medicine, side by side with that which we all recognise in surgery. The treatment of phthisis, the introduction of numerous synthetic chemical compounds as drugs in the treatment especially of nervous disorders, opotherapy, or the use of gland extracts, serum-therapy, the more elaborate use of baths and of the powers of light and electricity, may be mentioned as instances.

But, amongst ourselves, it is permissible to confess that very much still remains to be done, and that in very numerous cases of disease we have no means of directly checking or even influencing the process, but have to confine ourselves to watching and waiting, relieving artificially the various symptoms which arise, and guarding our patients as far as possible from the risk of serious complications.

Many as have been the triumphs of research in recent years into the nature of infectious diseases, we have still no means of *directly* treating the majority of the disorders comprised under this term. In a few instances we have specific remedies, such as quinine in malaria, mercury in syphilis, perhaps the salicylates in rheumatism, and possibly potassium iodide in actinomycosis. Bacteriological researches also have placed in our hands the possibility of neutralising, at least in part, the pernicious effects of diphtheria, tetanus, and septicæmia. And it is yet to be hoped that in the course of the next few years many other similar diseases will be brought into line with these, and that the antitoxins to their specific viri may be found and utilised.

Perhaps we must not complain if progress in this respect is slow. The treatment of syphilis and malaria has been known for years; the antitoxin treatment of the above diseases dates back only some dozen years; and, if other antitoxins can be added at the rate indicated until each of the infectious diseases has found its appropriate remedy, neither the profession nor the public ought to be dissatisfied.

But when we turn from general and infectious diseases to the various local conditions of disease, we are brought face to face with numerous pathological conditions, the direct treatment of which can scarcely be called triumphantly successful. If acquired pathological conditions mainly consist of inflammatory lesions, degenerative changes, and

the formation of new growths, in how few of these cases can we really check the progress or determine the cessation of the disease. Every organ or part of the body may be the subject of inflammation, and, with the exception of some few slight or extensive surface lesions, of which the causative micro-organisms may be dealt with by antiseptics, such inflammations must run their course, and find their end either in the natural death of the micro-organism; or in the power of the macro-organism to provide antitoxins or anti-bactericidal materials, or by mere sufficiency of resisting power to starve out the bacteria, and render their toxins innocuous. Such powers of resistance are no doubt largely assisted by well-judged treatment, consisting in different cases of improved hygienic conditions, methods for the relief of strain on the functions of an organ, and medicinal measures acting often in some not too obvious manner.

Under this head come the catarrhal inflammations in almost every part of the body, the inflammatory lesions of the lungs, of solid organs, of joints, bones, and skin. A striking instance of our helplessness, and of the little progress which has been made for a long series of years, seems to me to be given by acute inflammation of the kidney, whether this be called acute Bright's disease or acute nephritis. The immediate treatment of this complaint, that is, the treatment directed to allaying the inflammation and bringing about a return of the kidney to a healthy condition, has altered scarcely at all during the last seventy years.

The principles of treatment are still admitted to be the attempt to give rest to the kidney, by diverting its secretions or excretions to the bowel and to the skin, for which purpose diaphoretics and purgatives are in different forms administered. The extent to which this treatment does good is very difficult to estimate: the quite mild cases would get well of themselves, and the very severe cases are not apparently touched by the treatment. But even admitting that many cases are appreciably benefited by this line of treatment, it is again a method of watching, and waiting, and hoping; hoping that the disturbance which the micro-organism or toxin has produced in the organ will in due course subside. The interesting discussion at the British Medical Association in July last on "The Treatment of Chronic Renal Disease," * ably introduced by Dr. Hale White, illustrates the same posi-

* *Brit. Med. Journ.*, vol. ii, 1904, p. 886.

tion. So far as can be judged from the reports in the medical papers, the consideration of any direct means of dealing with the kidney itself took a subordinate place, and attention was chiefly directed to neutralising its effects by drugs or diet, the effects chiefly dealt with being loss of albumin, high tension and hypertrophied heart, obesity, œdema, and acute and chronic uræmia.

I am not unaware that in addition to the mechanical and physiological rest which the physician tries to impose, the relief of tension in the kidney has also been attempted and practised by the surgeon, by incision in acute stages and decapsulation in chronic, but these latter methods are no more directly curative in the proper sense of the term than the former. They can only place the organ presumably under more favourable conditions while the inflammation lasts, and may prevent the development of local changes of a prejudicial kind. Can it be shown that catarrhal lesions of the mucous membranes subside any more rapidly because there are no tense capsules to compress the swollen tissues?

In other parts of the body similar difficulties with regard to direct treatment may be recognised.

When we turn to degenerative processes it is obvious that little can be expected from curative methods, and that we must look rather to preventive measures to save us from disaster here. This is sufficiently manifest in the speeches in the discussion on arterio-sclerosis at the same meeting in July last.

The great difficulties in the treatment of the degenerative diseases of the nervous system are well known and illustrate this fact: even when these degenerations are presumably, or at least primarily, due to a more or less amenable poison, like syphilis, we still see, in locomotor ataxy and general paralysis of the insane, how little result can be obtained from the administration of antisyphilitic remedies, though recently improvements in locomotor ataxy have been claimed as the result of adequate and persistent treatment by potassium iodide. And even if in aneurysm the iodide of potassium has sometimes rendered great services, this has generally been attributed to its influence in promoting coagulation of blood within the sac rather than to any effect upon the primary lesion of the arterial walls.

I do not want to labour these points, and I only now bring them forward in order to suggest lines upon which

inquiries may proceed, and subjects upon which members of this Society may some day be prepared to enlighten their colleagues.

If I turn again to recent advances in therapeutics of which the profession may be proud, and which may be claimed to justify the existence of the profession, and the confidence of the public, I should like to suggest that the meetings of the Clinical Society would be well occupied sometimes in discussing the merits of several of these, which as far as my memory serves me, aided by a reference to the *Transactions*, have not occupied so large a place in our proceedings as their importance perhaps demands. Discussions of this kind upon methods of treatment which have captivated the imagination of the public or of the profession, sometimes on rather inadequate grounds, have occasionally, whether by a process of elevation or depression, brought these methods to their proper level.

I remember that some years ago a lively discussion took place, I think at this Society, upon the value of phosphorus in the treatment of leucocythæmia and allied diseases, with the result, as it seemed to me, of considerably damping the ardour of many who had been led to hope great things from this drug. At a sister society some twenty years ago a well-organised debate upon the treatment of typhoid fever by the cold bath appeared to me to transfer the cold water effects from the patients to the physicians, and the enthusiasm of the latter suffered the refrigeration, which had been intended for the bodies of the former. The treatment since that time has undergone varying fortunes: and though the general value of reduction of temperature is on all hands admitted, the inconveniences of the detailed method practised from 1874 to 1880 have been shown to be too great to allow of their universal application, even in hospital practice; and cold sponging, wet pack, ice cradles, and other methods of attaining the same end, are generally accepted as the limits of the practice.

But to return to more recent therapeutics, the following are some of the subjects on which, as far as I can ascertain, few papers or none have been read at the Clinical Society:—The Nauheim treatment of heart disease; Finsen's treatment by light; treatment by high-frequency currents, by Röntgen rays, and by radium; the therapeutical value of supra-renal products and allied substances. And in mentioning the name of

Finsen, I cannot forbear from expressing our sense of the loss which medical science has sustained in the death of this eminent man.

Doubtless many other subjects will suggest themselves to members, but it suffices for me to indicate what is in my mind. Difficulties may present themselves. It may be said that in the consideration of the value of a therapeutical method, the presentation of one or two cases seldom suffices to demonstrate even the probability of the influence of the method in question, and that large collections of cases with the necessary analyses and remarks, will not always fall within the scope of the kind of paper prescribed by the rules of the Society. My predecessor in the Presidential Chair, Professor Howard Marsh, whose recent induction into the Chair of Surgery at the University of Cambridge is a subject of gratification to all of us here, indeed to the profession in London, suggested in his presidential address two years ago that arrangements might with advantage be made for discussions on selected subjects, and for the analysis and estimation of the results of such discussions afterwards. I am not aware that any decided steps were taken to realise this project, but while feeling myself that the lines adopted by the Society hitherto, distinctive as they are in the insistence on individual cases as the basis of the paper presented, are the best, it appears to me feasible for the Council occasionally to allow a discussion on some such subject as those I have alluded to, to be introduced by a paper deviating in some respects from that which the Society specially favours.

I have already alluded to the eminent success, so far as numbers are concerned, of the four meetings in the Session at which living cases are exhibited. At these meetings from 70 to 100 members and visitors attend; nearly all the cases are thoroughly investigated, discussed, and re-discussed in the adjacent rooms, and valuable experience is gained, and lessons are learned. Especially the results of surgical operations, and manipulative treatment are exhibited, and rare cases of nervous disease become familiar to large numbers who might otherwise not see them. But the evenings are not always perfect. After the exhibition of cases, some 50 per cent. of those who have observed them leave, and the hour from nine to ten in which the cases are successively called out, and comments in public are invited, has often proved to be a rather gloomy sixty minutes. Upon many cases no comments

are made in open meeting; those who are in a position to criticise or approve have said their say in the exhibition rooms, and have gone away. The exhibitor has an opportunity, it is true, of supplementing his description or remarks, but such additions ought not to be necessary, and that is not the real object of the assembly during the hour named. I believe it is admitted by those who remember the first regulations under which the meetings reserved for exhibited cases were conducted that the present method is far more satisfactory. But the honorary secretaries, and some other members of the Society, as well as myself, have tried to imagine whether some improvement might not yet be possible, so that we might be spared the feeling that one half of the evening has been imperfectly utilised. It is possible that the bulk of the members do not share this sense of, shall I say, asthenia in the Society on these occasions, and from the last words of his address it is obvious that your former President did not. Moreover, until we are all reasonably agreed that a change is desirable, I would not urge one; all the less as a change which would maintain the essentials and meet the new requirements is certainly difficult to devise.

I think there is no doubt that in all respects, including the above, the Society, as the Report of the Council indicates, is successful, and is doing good work.

In closing this address I cannot help alluding to an event which must have a beneficial influence upon clinical medicine, and in which the Clinical Society has perhaps only by a few days missed having a share. I refer to the visit of the French physicians and surgeons to London. I hope it may be the first of many similar interchanges of national goodwill, and of knowledge and thought upon those medical subjects which we all have at heart.

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COMMUNICATIONS.

I.—*The Cure of Infantile Paralysis of the Shoulder by Nerve-grafting.* By WILFRED HARRIS, M.D., M.R.C.P., and V. WARREN LOW, M.D., F.R.C.S. *Read October* 14, 1904.

WE do not propose here to enter fully into the history of nerve-grafting further than to state that the possibility of operating on the nerves of the brachial plexus in cases of paralysis of a group of muscles mainly or entirely supplied by one of the primary nerves of the plexus was suggested to us by the beneficial results produced by Ballance, Kennedy, and others in cases of facial paralysis. This they effected by means of grafting the facial nerve on to the spinal accessory, or the hypoglossal nerve.

Kennedy also, in January, 1903, published some favourable results in cases of birth paralysis of one upper extremity of the Erb-Duchenne type, that is to say, paralysis of the deltoid, spinati, biceps, brachialis anticus, and supinator longus. His operation was not, strictly speaking, nerve-grafting, for he cut out a piece of the upper cord of the plexus formed by the combined fifth and sixth nerves, and sutured the central ends of these nerves to the divided supra-scapular nerve and the branches to the outer and posterior cords. Last year we read a paper at the Swansea meeting of the British Medical Association in which we detailed two cases of Erb's paralysis of the arm in adults, in which the conditions found at the operation by stimulation of the fifth and sixth cervical nerves

proved that the fifth nerve alone was damaged. We therefore came to the conclusion that the deltoid, spinati, and biceps received their whole motor supply from the fifth nerve, or from the fourth and fifth, and that the sixth nerve sent no motor nerves to these muscles. Acting on this hypothesis, we decided that there should be an equal probability of benefit from nerve-grafting in cases of infantile paralysis of the upper extremity in cases in which the paralysis was limited to the muscles supplied by one segment of the spinal cord, and therefore by the nerve entering the plexus from that segment. Moreover, we considered it possible that the motor fibres to the deltoid and to the spinati might be isolated from those supplying the biceps and brachialis anticus as they run side by side in the fifth cervical nerve, and each bundle dealt with separately. By splitting the fifth nerve longitudinally, so as to separate these groups of nerve-fibres, either bundle might then be cut across, and its peripheral end grafted into a neighbouring healthy nerve containing motor fibres, such as the sixth cervical nerve. Now, careful dissection of the fifth cervical nerve and of its junction with the sixth had shown one of us (W. H.) that the fifth nerve naturally divides into three bundles as it joins the sixth nerve, a posterior bundle, forming about half the volume of the nerve, and two anterior bundles, an upper and a lower, destined for the supra-scapular nerve and the outer cord respectively.

These bundles run side by side in the fifth nerve up to the foramen, and it therefore seemed practicable to split the nerve longitudinally into two portions, so as to separate off the supra-scapular bundle, together with the majority of the fibres of the posterior branch from the bundle to the outer cord and biceps. A suitable case for such an operation presented itself in a child who was at the time attending the electrical department at St. Mary's Hospital for infantile paralysis of the deltoid and spinati muscles of the right arm, which were wasting rapidly, and presented the complete reaction of degeneration, with no reaction whatever to faradism, even by a strong current, under an anæsthetic. The patient is a little girl, who, at the age of twenty months, suffered from a typical attack of infantile paralysis, affecting the right upper extremity. This was in February, 1903, and, though the whole arm was weak at first, all the muscles recovered below the shoulder, except for slight atrophy of the thenar eminence. The deltoid and the supra- and infra-

spinati remained completely paralysed, and rapidly wasted, so that when the child was first seen in May, 1903, these muscles were markedly wasted, and presented typical reaction of degeneration, with no reaction whatever to faradism.

Galvanism twice weekly for four weeks produced no change, not the slightest power of abduction of the shoulder being shown. After carefully considering the conditions, and explaining them to the mother, we determined to operate on the lines already indicated. This was done on July 8th, 1903. A vertical incision, about two and a half inches in length, was made along the posterior border of the right sterno-mastoid, reaching nearly to the clavicle. The external jugular vein was exposed and dragged aside, and the outer border of the scalenus anticus defined. The latter muscle was retracted, and, after exposing and cleaning the fifth and sixth cervical nerves, each nerve was first insulated by passing beneath it a bent vulcanite spatula, and then stimulated electrically by weak faradism.

On stimulating the fifth nerve, slight, though distinct, fibrillary contraction was seen in the deltoid, with powerful flexion of the elbow by the biceps. No contraction at all of the deltoid was seen on stimulating the sixth nerve, but there was slight movement of the biceps to this nerve, with powerful contraction of the pectoral, triceps, and latissimus. We therefore concluded that the lesion in the cord was in the fifth segment, and that the sixth nerve was, at any rate, sufficiently healthy to provide a good stock for the graft. Now, in this child the biceps was practically normal, and it was therefore imperative to divide only those bundles of the fifth nerve supplying the supra-scapular and circumflex nerves, leaving the bundle to the outer cord intact. The fifth nerve was therefore split longitudinally into two equal portions for a distance of about an inch by a Graefe's knife. Each portion was insulated and stimulated, when it was found that slight fibrillary contraction of the deltoid, but none at all of the biceps, followed stimulation of the upper portion, while vigorous flexion of the forearm occurred on stimulation of the lower. We felt, accordingly, that it was perfectly safe to divide the upper portion. This was done as near the foramen as possible, and its peripheral end turned over in front of the uncut lower portion of the fifth nerve, and stitched into a small nick made in the anterior aspect of the sixth nerve. The wound was closed, and the child returned to bed, and the head kept between sandbags. On

examination the next day the child was able to flex the forearm as before. The recovery from the operation was uneventful. No change in the condition of the paralysis was observed until the following January, six months after the operation, and eleven months after the onset of the paralysis. Slight power of abduction of the shoulder was then first noticed. Progress, at first slight, has during the last three months increased in rapidity, and she is now able to hold the arm out at right angles from the shoulder, and to raise the arm over the head.

Under an anæsthetic, ten days ago, slight, though distinct, reaction to faradism was obtained in the deltoid, and we have every hope that in this case complete, or nearly complete, use of the deltoid will be regained.

In every such case a most essential part of the procedure is the careful stimulation of the exposed nerves, as, without this, it is impossible to be sure of the motor distribution of the nerves entering the plexus.

In a recent paper one of us (W. H.) has shown that the variations in the comparative size of the fifth cervical nerve may be considerable, according as the plexus is of the high or low type, prefixed or post-fixed. In some cases of high plexus the fifth nerve may be nearly as large as the sixth, while in others it may be as small as one fourth the size of the sixth nerve. One of us (W. H.) has recently, through the kindness of Mr. C. H. Fagge, had the opportunity of seeing an operation on the upper nerves of the plexus in a case of birth paralysis at the Evelina Hospital. In this case the fifth nerve was the smallest he has yet seen, certainly not larger than one sixth part of the sixth nerve. Indeed, the fifth nerve was so small that we could not at first recognise it, as it appeared to be an abnormal branch from the phrenic, which it scarcely exceeded in size. Mr. Fagge stimulated the fifth and sixth nerves successively, and rather stronger contractions of the deltoid were obtained from the sixth than from the fifth nerve. In the low, or post-fixed, type of plexus it is therefore clear that the motor supply of the deltoid is at least equally as great from the sixth nerve as from the fifth. Although a shrewd guess as to whether the plexus is high or low in type may be made by comparing the size of the fifth and sixth nerves when exposed at the operation, yet no published list of motor root supply can possibly replace the certain knowledge gained by careful stimulation of the nerves at the time of the operation, and it is according

to these results that a decision must at once be made as to the particular form of nerve-grafting suitable to the individual case.

The application of this operation is necessarily limited, owing to the comparative rarity of such a localised infantile paralysis, though in certain injuries of the plexus and birth palsies it probably may be of service. Its value, however, is obvious when the almost absolute uselessness of an arm with a paralysed deltoid is realised, such as was the case in the child shown here to-night.

II.—*Cases of Inflammation in and around the Liver of Secondary Origin.* By W. G. Spencer. *Read October* 14, 1904.

I PROPOSE to refer to some cases which can be, with reasonable probability, excluded from the common categories of cirrhosis of the liver and ascites, although it may be that such cases have been often in the past somewhat loosely classified as alcoholic, syphilitic, or biliary in origin.

A resort to surgical exploration allows of a direct examination of the disease at a much earlier stage than that reached when the examination is made after death from it. It is from this latter that knowledge has been hitherto mainly drawn when perhaps affections having widely different origins come to resemble one another in a common ending. An early surgical exploration of such cases as those described below is the more worthy of favourable consideration, seeing that an arrest of the inflammatory process tends to follow the operation. For it appears to bring about a restoration of the equilibrium normally existing between exudation and absorption, which in the affected region had become disturbed.

Case 1. *An enlargement of the liver simulating hydatids which subsided after incision.*—A male lodging-house keeper æt. 41 had always lived in Pimlico, and had been a total abstainer from alcohol; there was no evidence at all that he had ever had syphilis. There was nothing noteworthy in his family history. Twenty-two years ago he had typhoid fever; eight or nine years ago he had an acute attack of pain in the epigastric region, which lasted fourteen days. Three years ago he was laid up for six weeks with muscular rheumatism. Otherwise he had been in good health. In the first week of September, 1903, a cold wet season, three and a half months before the operation, he was at Deal on a holiday, when he was seized with a stabbing pain under the ribs in the right hypochondrium, which extended inwards towards the epigastrium, and caused his breathing to become shallow, and even difficult and painful. It pained him especially when he sat down, and when he rose up from sitting. The pain was at

first continuous so that he could not sleep at night. After a week the pain subsided to be of a dull aching character, placed rather above and nearer to the middle line than had been the primary seat, more directly below the xiphoid cartilage, and his breathing then became easier. From Deal he went to Dover, where he stayed for eleven weeks. He was for part of the time under Dr. Ormsby, and was meanwhile slightly jaundiced, his face and hands appearing somewhat yellow. For three weeks he felt better, his appetite was good, his bowels regular, and he thought he put on flesh. He then came to London, but as he did not feel well, and the upper part of the abdomen was swollen; he attended the French Hospital under Dr. George Ogilvie. From the physical signs, and also by a process of exclusion, Dr. Ogilvie, in conjunction with Mr. Edmund Owen, diagnosed hydatid disease of the liver, especially affecting the right lobe.

The patient thereupon sought admission to the Westminster Hospital under Dr. de Havilland Hall. There was then found occupying the left hypochondrium, the epigastrium, and right hypochondrium, a firmly resisting mass which extended down nearly to the umbilicus. The free margin below had the character of an enlarged liver, and moved up and down with respiration. The upper limit, as indicated by dulness to percussion, was marked by the fourth intercostal space in the left nipple line and the fifth rib on the right side. The apex-beat was most marked just external to and level with the left nipple. The mass, as felt below the ribs, was smooth and even, firmly elastic, painless to pressure except for a spot just below the xiphoid cartilage, which was tender upon deep palpation. It was uniformly dull to percussion. Behind there was no increased dulness. At the base of the left lung were a few fine crepitations.

An X-ray photograph showed a uniform shadow over the area marked out by percussion, including the slight displacement of the heart outwards. For two or three days shortly after admission the temperature was 100° F., or a little above, at night. He weighed 10 st. 4 lbs., not having during his illness lost weight to any great extent. The blood was normal, except that the hæmoglobin was estimated at 86 per cent. Nothing else abnormal was found, and for the fortnight the patient was under observation there was no marked change. I examined the case with Dr. Hall, and it then seemed as if the most prominent part of the swelling was in the left hypochondrium behind the upper part of the

left rectus; also the intercostal spaces on the left side felt more tense than on the right. These observations appeared to be confirmed under the anæsthetic, and account for the position of the incision.

On December 22, 1903, I explored the swelling in the presence of Drs. Hall, Ogilvie, and Ormsby, and Mr. J. M. Barlet, the House Surgeon at the French Hospital. The incision was made through the upper part of the left linea semilunaris. The subperitoneal tissue was thickened and excessively vascular, venous blood flowing freely. On dividing the peritoneum it was found to be united to the liver by recent very vascular inflammatory tissue continuous with the capsule of the liver, falciform ligament, and diaphragm. Expecting to find a hydatid cyst, I pushed the finger for about an inch into the liver substance, but the bleeding became free, and there was no sign of a cyst-wall, so I was brought to a standstill, and had to plug the hole in the liver firmly with iodoform gauze. The incision was thereupon extended downwards until the finger could be passed into the abdominal cavity, where the enlargement of the liver was found to be uniform. Both the upper and under parts of the liver felt smoothly elastic; there was no sign of any tense bulging cyst, nor of any new growth; nothing was felt except soft adhesions between the anterior aspect and the abdominal wall, which were not further disturbed. The upper part of the abdomen otherwise felt normal. The abdominal wound was then closed except for the plug. Following the operation the temperature fell to 97° F., next it rose to 101·8°, and afterwards remained normal. There was free oozing of blood from the wound, which continued in spite of firm pressure on the plug. On the 8th day after the operation the bleeding became more severe, the pulse rose to 160, and the temperature fell to 97·4°. In my absence Mr. Paton swabbed out the wound with turpentine and replugged it. During the second week blood, mixed with some pus, continued to escape, and the patient became distinctly anæmic, whilst the enlargement of the liver seemed stationary. Three weeks after the operation the patient had clearly wasted whilst the enlargement of the liver persisted, but the bleeding from the wound had ceased. His mouth and lips were then furred and dry, his gums bled, and the hæmoglobin in the blood was estimated to be 60 per cent. After three weeks from the exploration the patient rapidly improved in all respects; the liver, as evidenced by percussion, got rapidly

smaller until it projected hardly at all below the ribs. The wound healed, and he was able to get up. Six weeks after the exploration his weight was 8 st. 12 lbs., *i. e.* a loss of more than a stone after the operation. Three weeks later he had regained this weight, and appeared to be returning to good health when he left for a convalescent home. Four months after the operation he was in good health, weighing eleven stone, *i. e.* one stone more than he weighed before the operation; nothing could be found of the liver below the ribs. Quite lately a letter was received saying he was in excellent health.

In this case before the exploration, a diagnosis of hydatid cysts of the liver seemed almost assured; following the exploration, primary malignant disease of the liver appeared a probable view. We may now think, perhaps, that the patient was suffering from hepatitis, and peri-hepatitis as the result of a chill acquired whilst on his holiday at Deal.

Case 2. *Enlargement of the liver, complicated by jaundice, acute fever, and rigors, relieved by drainage of the gall-bladder.*— A dressmaker, single, æt. 43, had never taken any alcohol regularly, nor to excess. There was no evidence of syphilis. She had suffered from rheumatism five years before, and a year previously had been treated for gastric ulcer. During the past year she had had three slight attacks, and then a fourth attack occurred of a more severe character about a fortnight before admission to the Westminster Hospital. As she was going to her work one morning she began to feel sick and took some hot water. She thereupon vomited a quantity of frothy, bitter, bad-smelling fluid. Four days later the patient noticed that she was becoming jaundiced, from which she had never suffered before.

The patient was admitted under Dr. Hall with deep jaundice, the tongue furred, the appetite poor, the bowels constipated, the urine containing bile, but the pulse and temperature were normal. The liver was found to be enlarged, reaching below the umbilicus, the right lobe to the crest of the ilium. On the fourth day after admission the patient seemed doing well, the jaundice less, the liver smaller, and the bowels had been freely opened by enemata. She weighed 6 st. 13 lbs. On the next day the temperature rose above 100°, the pulse to 108, the jaundice deepened, and she vomited. On the 7th day she had a severe rigor, followed by profuse sweating, the temperature rising to 106·2° F., with

repeated vomitings, and deep jaundice, and the pulse-rate was 132. The rigors were repeated the next day, there was persistent vomiting, the temperature fell between the rigors to 98·8°, the pulse was counted several times at about 160, and she became very weak. A severe herpes labialis commenced. Dr. Hall and I agreed that the patient had septic pyelophlebitis, and I undertook drainage of the gall-bladder. I made an incision in the right linea semilunaris long enough to expose the liver, which reached to the crest of the ilium, and the liver was then lifted up in order to get at the gall-bladder. The liver was uniformly enlarged, smooth, and elastic, bile-stained, but not more so than the other tissues. There were some adhesions between the gall-bladder and the first part of the duodenum, which, being separated, exposed a small, nearly empty, gall-bladder, not altered except by inflammatory thickening around it. There was no sign of obstruction nor of calculus either in the gall-bladder and cystic duct on the one hand, or in the common duct and head of the pancreas on the other; also the pylorus and adjacent portion of the stomach seemed normal except for adhesions. A glass tube was tied into the gall-bladder and connected with a receiver externally. Bile flowed freely, but when examined no pus, nor sediment, nor special organisms were found in it. The temperature after the operation did not rise above normal, and the pulse slowly diminished in frequency. The jaundice gradually disappeared, and the motions became well coloured, whilst bile was still escaping freely, showing that there was no obstruction to its flow by way of the common duct. The tube remained *in situ* for about three weeks, after which the biliary fistula closed rapidly. The patient put on flesh, and the liver shrunk so as not to appear but little below the ribs. She was sent to a convalescent home about seven weeks after the operation. Three months after the operation she had put on much flesh, and Dr. Abrahams found the edge of the liver only just below the ribs.

The inflammatory condition seems to have been situated in the liver, and the acute symptoms and rigors to have been caused by extension to the hepatic veins from the stomach. All this must have been due to recent inflammatory exudation, for the swollen liver shrank so very rapidly. Presumably the free escape of bile by the tube relieved tension, but there was no sign of any pus draining off by the tube. She had evidently suffered beforehand from chronic gastric

ulceration which had set up some peritonitis, and it seems most probable that extension took place from the stomach by way of the veins to the liver.

I have had occasion recently to operate upon other cases formerly classed as cirrhosis and ascites in which all syphilitic and alcoholic history was either absent or of the most indefinite kind. Two of them will be briefly mentioned, as they have been elsewhere described.

Case 3. *Chronic peritonitis around the spleen and liver, with ascites and anæmia.*—Under Dr. Murrell. See *Lancet*, 1900, i, June 16, and *Medical Press and Circular*, 1902, i, January 8.

Ultimately this patient was termed an instance of Banti's disease, but the indefinite way in which this name seems to be used appears to indicate that the term has not been given to a sharply defined disease.

A girl, æt. 19, was admitted to the Westminster Hospital in November, 1899, with anæmia (3,830,000 red corpuscles per 1 c.mm.), œdema, amenorrhœa without pregnancy, no leucorrhœa nor albuminuria. There was then no ascites, and the colourless corpuscles were 3000 per 1 c.mm., chiefly lymphocytes and finely granular oxyphile polynuclear leucocytes. She remained in the hospital about a month, during which the anæmia was markedly benefited by iron. Three weeks after leaving the hospital (January 9, 1900) she was readmitted, having in the meantime suffered from a bad cough and sore throat. Her temperature was then 104° F., the urine contained much blood and albumen without casts, there was a soft apical systolic murmur, and there was a large quantity of ascitic fluid in the peritoneal cavity. The red blood corpuscles were about the same as before, 3,450,000 per 1 c.mm., the white corpuscles 7000 per 1 c.mm., 54 per cent. being fine granular oxyphile cells, most of the rest resembling lymphocytes. The ascitic fluid continued to increase and to enfeeble the heart.

On January 27, about three weeks after the second admission, I washed out and drained the abdomen below the umbilicus, keeping the glass tube in for about a week. It was then found that the spleen was enlarged, its edge extending two inches below the ribs. The fluid soon re-accumulated. A month later, February 26, I made an epigastric incision, and fixed the omentum in the wound under the skin. At the time the enlarged spleen was felt

also there were signs of thickening of its capsule and of the surface of the liver. The patient now rapidly improved. She was discharged on April 19, about two months after the fixation of the omentum. On June 1 she stated that she felt perfectly well and able to take up her occupation. The cardiac murmur had practically disappeared; there were some enlarged superficial abdominal veins. Sixteen months later she was admitted for the third time with re-accumulation of fluid and a temperature of 102·3° F., all attributed to a fresh cold. The abdomen was again drained, but she died about a month later.

Post mortem adhesions were found between the liver, spleen, diaphragm, and abdominal wall. The peritoneum was generally thickened. The omentum was firmly fixed in the abdominal wall, and it contained large communicating vessels. The liver showed an increase in interlobular fibrous tissue; the spleen was enlarged, reddened, with an infarct of old standing, also smaller ones breaking down. There was slight mitral disease.

It seems most probable that infection through the lungs was the cause of the inflammation around and in the liver, also of the ascites, and enlargement of the spleen; this was relieved for a year by the union of the omentum with the abdominal wall, death following another attack through the lungs.

Case 4. *Peritonitis around the liver, with ascites, commencing after puerperal fever.*—In this patient, likewise, there appeared as the chief lesion inflammation over the surface of the liver and the neighbouring region. The patient, æt. 30, was in good health until after a labour, which was followed by some form of puerperal fever, and this by ascites and mitral disease. In the course of two years she was tapped ten times in the Westminster Hospital. When Dr. Hall asked me to operate, the abdomen was much distended with fluid, there was some œdema of the ankles, and a marked systolic murmur at the cardiac apex. I washed out and drained the abdomen through a hypogastric incision, and sutured the omentum into the abdominal wall in the epigastrium. The patient was shown nine months later at the Medical Society, when she expressed herself as feeling well and able to attend to her house. There was then no ascites, nor œdema of the ankles, and it was a question whether any abnormal heart sound could be heard. The examination of

the peritoneum at the time of the operation appeared to me to show chronic peritonitis, especially in the neighbourhood of the liver, and it further seems probable that this was started by the puerperal fever.

Conclusion.—I have in a previous paper referred to four cases of syphilitic disease of the liver benefited by surgical operation (*Clin. Soc. Trans.*, 1899, vol. xxxii, p. 46). The influence of syphilis and alcohol can be, I believe, excluded in the four cases I now describe. None of them showed biliary disease, none had suffered from malaria, dysentery, typhoid fever, nor pyæmia.

Of the four cases detailed, in the first the disease seems to have been started by a chill over the liver, the second by septic absorption following gastric ulceration, the third by way of the lungs from some influenzal, perchance pneumococcal, infection, and the fourth by some mild septic absorption from the puerperal uterus.

But, although of different origin, yet the effect was an inflammation, either in the liver causing rapid enlargement, or around the liver in the peritoneum causing exudation. It follows, as a general observation, that one should not hastily label a case "cirrhosis and ascites" and give its causation no further consideration, nor presume for it of necessity alcoholic or syphilitic antecedents. Surgical exploration, with the establishment of the venous anastomosis connected with the names of Talma and Morrison, gives renewed vigour and interest to the attempts to differentiate cases hitherto included under the same name.

III.—*Congenital Elevation of the Scapula.* By Charles R. Keyser, F.R.C.S. *Read November* 11, 1904.

JUDGING from the number of recorded cases this deformity is rare, and as I have recently had the opportunity of observing one, I have put together a few facts concerning this malformation in the form of a paper in the hope that it may prove of interest to the members of this Society.

The patient was shown at the Clinical Evening held by this Society on January 22. The details of the case are briefly as follows:

Grace J., æt. 9, was brought to Hospital on December 17, 1903, because of deformity of the left shoulder-blade, which had been noticed one month. There was no history of accident or infantile paralysis. No other member of the family has a similar or any other deformity. The patient has always been "delicate," but has no definite symptoms.

On examination the left scapula is 2½ inches higher than the right, but the bone is of the same size as its fellow. Movement of the arm, both active and passive, is normal. There is no osseous bridge to be felt between the vertebral border of the scapula and the spine, nor is there any lateral curvature of the spine. The spinous process of the third dorsal vertebra, however, feels thicker than normal, and is apparently displaced slightly to the left. The inferior angle of the scapula is on a level with the interval between the sixth and seventh dorsal spines. There is no paralysis of any muscle. X rays show nothing abnormal, and the two arms are of the same length. The child's head is ricketty in appearance, but there are no other signs of rickets, nor any other deformity. The teeth are slightly notched and ridged, as is commonly the case in ill-nourished children.

As the child suffered absolutely no pain or inconvenience from her condition no active treatment has been adopted, especially as the deformity is barely visible, except when the patient is undressed. In the only other similar case with which I am acquainted where operative measures were adopted in order to correct the deformity, very little, if any, benefit followed a very severe operation.

Cases of congenital elevation of the scapula may be classified into four groups.

1. Where there is a bridge of bone between the vertebral border of the scapula and the spine.
2. Where there is absolute absence of one or more muscles forming the shoulder-girdle.
3. Where there is a long and everted supra-spinous portion of the scapula.
4. Where there is no osseous bridge, and the scapula is normal or smaller than usual, with short or otherwise defective muscles.

The great majority of recorded cases fall under the last group.

Group 2 is very rare, only five instances having been met with.

The most interesting cases are those where there is an osseous bridge between the scapula and the spine, as it is still undecided how this piece of bone is formed. Moreover it can be dealt with surgically with most gratifying results. When present it is usually triangular in shape, the base being attached to the vertebral border of the scapula, and the apex to the spinous process of either the seventh cervical or the first dorsal vertebra. In many instances the actual union to the scapula and spine is by cartilage which allows a certain amount of movement. The arm, however, can rarely be raised beyond a right angle. Various theories have been brought forward to explain the presence of this abnormal bridge of bone, the chief of which are that it is formed in connection with the vertebræ, and afterwards becomes attached to the scapula, or that the reverse of this is the case, or that it is independent of both.

Messrs. Willett and Walsham described the first two cases, records of which are to be found in the *Medico-Chirurgical Transactions* for 1880 and 1883 respectively.

In the first of these there were multiple deformities present besides that of the scapula. The clavicles, ribs, and sternum were all abnormal, and besides this there were twelve and a half vertebræ missing. The left scapula, which was fixed by a broad ridge of bone to the spine, was much shorter than normal, and was in appearance more like a scapula at birth. The osseous bridge was partially united by cartilage to the scapula.

The second case was that of a girl æt. 8, whose left scapula was one inch higher than the right. A triangular bridge of bone between the scapula and the seventh cervical vertebra was present, which caused much impairment of move-

ment of the arm. The osseous bridge was removed and was found to be connected to both the spine and scapula by a layer of cartilage. Similar cases have been recorded by Messrs. Goldthwaite and Painter (*Transactions of the American Orthopædic Association,* 1901), by H. S. Collier (*Reports of the Society for the Study of Disease in Children,* 1902), and by Messrs. Hibbs and Loewenstein (*New York Medical Record,* August 1st, 1903).

In discussing the formation of this osseous bridge Messrs. Willett and Walsham regarded it as an "abnormal development of the supra-scapular epiphysis which normally exists as a narrow ridge of bone along the posterior border of the scapula, and consequently as homologous to the supra-scapular bone of some of the lower vertebrates. The supra-scapular epiphysis in man may actually present an abnormal backward development comparable to that, say, of the rabbit. It is advancing, therefore, but one step further to compare the bridge of bone in our specimens to the distinct supra-scapula of the frog, and but one step further still to conceive it united to the spine as in the skate. This means that the shoulder-girth in these two cases has reverted to that of a lower type."

Cases similar to my case, where no osseous bridge is present, have been recorded by H. B. Robinson (*Reports of the Society for the Study of Disease in Children,* 1902), by Goldthwaite and Painter, and by Hibbs and Loewenstein.

It has been noticed that the deformity is unilateral and is more often present on the left side than on the right. The neck muscles are usually shortened, and other congenital malformations, such as spina bifida, shortness of the arm on the affected side, etc., are frequently present. The deformity has been said by some to be caused by either intra-uterine rickets, accident at birth, or an intra-uterine anterior poliomyelitis. The theory, however, which seems to account for the elevation of the shoulder in the most satisfactory manner is that suggested by Sprengel in 1891. He came to the conclusion that the arm became fixed, *in utero,* behind the back, and thereby caused elevation of the scapula.

IV.—*A Case of Traumatic Axillary Aneurysm successfully treated by Ligature of the First Part of the Axillary Artery after Extravasation had occurred from the Sac.*

By PHILIP D. TURNER, M.D., and RAYMOND JOHNSON, B.S. *Read November* 11, 1904.

MRS. T., æt. 70, tripped and fell down a flight of five or six stairs in November, 1899. She felt something "give way and return with a click" in the left shoulder, and experienced acute pain. From the moment of the injury she was unable to move the limb. A medical man who was summoned found that the forearm and hand were paralysed, and concluded that the brachial plexus had been injured; there was much stiffness with bruising of the forearm. Three weeks later the patient returned from Brighton, where the accident happened, to her home at Ryde, and was then first seen by Dr. Turner. At this date there was still almost complete paralysis of the muscles of the forearm and hand. In the anterior part of the axilla a small swelling could be felt under the pectoralis major; it was about the size of a small tangerine orange, but was not very clearly defined. The tumour was the seat of expansile pulsation, and a loud blowing murmur could be heard over it.

The pulse at the wrist was not perceptible; the limb was cold, and the general circulation feeble.

After the application of massage, electricity, and passive movement the paralysis of the forearm gradually passed off, but the muscles of the thenar and hypothenar eminences remained paralysed, and much stiffness persisted in the joints of the thumb. The circulation in the limb gradually improved, and after the lapse of several months the pulse at the wrist again became perceptible. The improvement in the muscular power was very gradual, and continued during a period of about two years.

During the first few months after the injury the axillary tumour, which was clearly a traumatic aneurysm of the axillary artery, increased in size, and by the sixth month was as large as a tennis ball. At times it appeared to be much more solid, and after the first four months attained a degree of hardness, which seemed to justify the hope that a consider-

able deposit of laminated clot had formed in it; the pulsation and bruit were, however, always more or less evident.

Gradually, during the third year after the accident, the aneurysm began to enlarge in a backward and downward direction, so that a finger could no longer be passed between it and the posterior axillary fold, and pulsation became evident on both the anterior and posterior aspects of the shoulder. This increase in the size of the aneurysm was not accompanied by pain or by any alteration in the extent of the paralysis, whilst the free circulation in the limb indicated that the collateral supply was well established.

In the earlier periods of the case, the question of operative interference seemed to be negatived by the stationary condition of the aneurysm, and the fact that some degree of consolidation appeared to have occurred in it. When the aneurysm began to increase in size the advisability of operation was discussed, but was not pressed, in view of the patient's age and her objection to any interference.

In October, 1902, nearly three years after the accident, Mrs. T— went again to stay at Brighton, and while there an extravasation, like a small bruise, appeared over the posterior axillary fold. A few days later she was seized with intense pain in this region, and a swelling as large as the fist appeared in the posterior part of the axilla, the skin over which became extensively discoloured by extravasation of blood beneath and into it.

On October 28 the patient was removed to her home at Ryde, and on the following day an attempt was made to ligature the third part of the subclavian artery through the usual incision. It was found, however, that the shoulder had been pushed upwards to such an extent by the aneurysm that the artery lay at least two and a half inches below the upper border of the clavicle, and the operation was abandoned. Two days later another extensive extravasation occurred into the axilla, so that the whole mass of effused blood formed a tumour as large as a fœtal head.

The condition was evidently one of extreme gravity, and called for immediate interference. The choice seemed to lie between the application of a ligature to the axillary artery below the clavicle; and the opening up of the axilla, and, after the evacuation of the extravasated blood, the ligature of the vessel above and below the origin of the aneurysm. It was decided to adopt the former course, and the operation was performed on November 2. Whilst the final cleansing of

the part was being carried out under the anæsthetic it was found that blood had just begun to ooze through a small opening in the skin; the hollow of the axilla was distended into a tense prominence, and the leakage was taking place in the deep groove between this and the chest wall. The artery was ligatured through an incision below the clavicle in the line of the vessel. After dividing the exposed fibres of the pectoralis major and the costo-coracoid membrane, the artery was reached at a considerable depth, and a silk ligature passed around it. On tightening the ligature all pulsation in the axilla at once ceased, but there was never at any time after the operation any anxiety as to the condition of the limb, the circulation in which was, from the first, quite satisfactory.

On the fifth day after the operation an opening formed in the skin of the inner wall of the axilla, in the position from which the slight escape of blood had already occurred; it was two and a half inches long, and had almost the appearance of an incised wound. From this opening a small quantity of blood continued to escape until about three weeks after the operation, when a free escape occurred and continued for three days. The opening was therefore slightly enlarged, and a large quantity of clot removed from the axillary cavity with the finger, which could be passed forward to the deep surface of the pectoralis major. From this time the discharge rapidly became reduced to a small quantity of blood-stained fluid, which later became purulent.

By Christmas all the swelling had subsided, and the contour of the shoulder was normal. The healing of the axillary opening was complete at the end of February, but a small sinus in the position of the drainage-tube inserted into the wound, through which the axillary artery was tied, was not complete until the middle of March. The patient's condition has, up to the present time, remained in every respect satisfactory.

The case is recorded as illustrating the successful treatment of a traumatic aneurysm, after extensive extravasation had occurred, by the application of a proximal ligature. That the ligature was applied to the axillary artery in the first part of its course instead of to the subclavian vessel is a matter of interest, but not of primary importance. The point in the vessel which was finally selected was only chosen because the application of the ligature to the third part of

the subclavian artery was abandoned on account of the extreme depth at which the vessel was situated.

At the time of the operation such extensive extravasation had occurred from the aneurysm that it was with considerable hesitation decided to adopt the proximal ligature in place of the much more severe measure of opening up the axilla and dealing with the artery at the site of the aneurysm. According to the usual teaching on this subject, the adoption of the proximal ligature when extensive diffusion has occurred is attended with but a small measure of success. One of the chief dangers is, no doubt, the increased risk of gangrene resulting from the interference with the collateral circulation caused by the distension of the tissues around the aneurysm with blood. In this case the long previous existence of the aneurysm had so opened up the collateral supply that even after extravasation had occurred the supply of blood to the limb remained good, and again, in the case of the upper limb the risk of gangrene in all interferences with the main vessels is undoubtedly less than in the lower limb. The successful result in this case was probably assisted by the spontaneous opening which occurred in the skin of the axilla, and through which a large quantity of the effused blood was quickly evacuated.

The treatment adopted in a single case is not necessarily justified by a successful result; still less does it follow that its adoption in other apparently similar cases would be equally fortunate.

It is interesting, however, to note that very striking cases are on record to show that even in apparently most unpromising circumstances the application of the proximal ligature may prove successful.

We may especially refer to a series of cases recorded by Major Birt, R.A.M.C. (*British Medical Journal*, March 15, 1902), all, except one, being instances of gunshot injuries, and treated in South Africa. Of these cases two are of especial interest in the present connection; they illustrate the successful treatment of extensive extravasation into the axilla after gunshot wound by ligature of the subclavian artery. In the first case a bullet wound of the axilla was rapidly followed by great extravasation in the axilla, with loss of the radial pulse and paralysis of the limb. On the sixth day, the swelling having rapidly increased, the third part of the subclavian artery was tied. The intense pain

was at once relieved, and the result was in every respect successful.

In the second case, also one of bullet wound of the axilla, the primary hæmorrhage, which threatened to prove fatal, was controlled by compression of the subclavian artery. Forty-eight hours later the axilla was occupied by a very extensive extravasation, the radial pulse was absent, the whole limb was paralysed and anæsthetic, and there was great pain. The third part of the subclavian was tied. By the eighth day the tumour was reduced in size, and muscular power and sensation were returning, and when the patient was invalided home he was making rapid progress towards a complete recovery.

V.—*Aneurysm of the Intra-cranial Part of the Left Internal Carotid Artery caused by a Bullet-Wound through the Right Eye.*

By H. Betham Robinson and Edred M. Corner. *Read November* 11, 1904.

THE history of this fatal case is brought forward on account of the rareness of the lesion and the imperfections in our knowledge of diagnosing such a condition. The sequence of symptoms was very carefully noted, and through the aid of Mr. Lawford and Mr. J. H. Fisher, we are able to append fairly complete ophthalmological notes.

A girl, æt. 18, was shot by a revolver bullet in the head immediately before admission into St. Thomas's Hospital. On examination she was completely unconscious, corneal reflexes absent, pupils small and reacting sluggishly to light, no paralysis, and general reflexes normal; pulse 108. Patient lay curled up in bed, occasionally moving her limbs, and resenting interference.

The point of entry of the bullet was about the right outer canthus, just below the eyebrow; the outer wall of the orbit was pierced, and the globe destroyed. The wound was cleaned up and the eye excised. There was no wound of exit.

On the following afternoon she regained consciousness and answered questions; in the evening she again became unconscious. She had retention of urine. On the third day she remained unconscious with flexed limbs and vomited for the first time; her temperature was 99·0° and her pulse 76. On the seventh day she was more sensible and cried a little; the left eye was suffused, and the cornea dull. A skiagram showed fragments of bullet, apparently between the cerebral hemispheres. The second bullet, which had entered the neck, was also demonstrated. On the ninth day the wound was suppurating; the patient was conscious and rational. The temperature was not above 99·0°, and the pulse was 80. On the eleventh day there was severe epistaxis, controlled by plugging the nares. On the twelfth day a shallow, nearly central ulcer, was noticed on the left cornea, which Mr. Lawford regarded as evidence of fifth nerve lesion, but this was the only sign of such. On the thirteenth day she had another and more severe attack of epistaxis, "losing quite a pint of blood

and becoming anæmic"; it was controlled by plugging. On the fifteenth day there was some discharge of clear fluid from the nose. On the twentieth day the corneal ulcer was rather larger and the vision poor; ung. atropin and bandage used. On the twenty-fourth day sensation was absent over almost the whole distribution of the upper two divisions of the fifth nerve. Ptosis and paralysis of the external rectus noted; vision rather better. On the thirty-third day there was complete external ophthalmoplegia; the area of anæsthesia was unchanged; general condition good, except for anæmia. Up to the forty-seventh day there was slow improvement, but the vision was gradually failing. On the forty-ninth day there was anæsthesia over the whole of the left arm as far as the elbow, and on the left side of the chest almost to the nipple. The knee-jerks were brisk; plantar reflexes normal. On the fifty-fourth day anæsthesia was present in the areas supplied by the supra-sternal, supra-clavicular, and transverse cervical nerves. On the fifty-ninth day there was no alteration in the anæsthetic areas, and vision remained the same. On the sixty-second day there was another sharp attack of hæmorrhage, which ceased spontaneously. Sixty-fifth day—Two more attacks of hæmorrhage, a considerable quantity of blood being lost. Sixty-seventh day—Movements of eye improved; ptosis less marked. Area of anæsthesia on face slightly lessened. External rectus still completely paralysed. Eighty-third day—Sensation returning in arm, neck, and chest. The conjunctival reflex was now obtained, and the corneal ulcer was clearing up. Eighty-fifth to ninety-fifth days—Frequent slight epistaxis. Movements of eye still improving. General condition fairly good. Examination of the nose and naso-pharynx by one of us (H. B. R.) gave no indication of the source of the blood, or of the course of the bullet. On the 112th and 113th days the bleeding was severe, and she vomited much blood; the hæmorrhage, which up to this time had been thought to come from the cavernous sinus, from its severity, frequent recurrences, and colour, apparently was of arterial origin. After a consultation with Mr. Pitts, in consideration that the bullet had destroyed the right eye, and the hæmorrhage came from the right nostril, it was agreed to ligature the right internal carotid and at the same time to put a ligature on the right external carotid to interfere with the formation of a collateral circulation through that channel. (Mr. Pitts, *St. Thomas's Hospital Reports*, vol. xii, p. 131.) This was accordingly done by one of us (E. M. C.)

Two slight hæmorrhages occurred within the first five days

of this operation. On the sixth day she awoke in the morning quite blind. Mr. J. H. Fisher examined her and reported as follows: "I see no fundus change. Optic disc rather pale and grey in colour. No evidence of embolism of the central artery of the retina. The defect in vision must be due to some lesion of the optic nerve behind the entrance of the central vessels, *i.e.* near the optic foramen. The suddenness of the onset suggests a hæmorrhage into the sheath of the optic nerve or the presence of blood outside. There are no signs of hæmorrhage into the orbit. The inactive pupil seems an old condition; if new, it excludes damage to the cortical visual centre, accounting for the sudden failure of vision." Ten days later, the 130th day, Mr. Lawford agreed with Mr. Fisher's report, adding that "the atrophic ulceration of the cornea was chiefly in the lower part; the cornea was insensitive; some chemosis present; the left eye was considerably proptosed." The patient had a transient hemiplegia on the left side. From this date there was no further hæmorrhage, and her general condition gradually improved; the partial paralysis passed off and consciousness returned. Her corneal ulceration slowly got worse, and she became totally blind. On the 195th day she was sent to a convalescent home, being quite well and cheerful, no bleeding having occurred since the 118th day, *i.e.* for seventy-seven days.

This period of improvement lasted for another twenty-six days. She was then readmitted for another severe hæmorrhage from the right nostril, which was stopped by plugging the nares. Three days later a very violent hæmorrhage occurred and she died in three minutes.

The autopsy was made by Dr. Singer, who reported as follows: "The bullet had pierced the inner wall of the right orbit, had traversed the body of the sphenoid, and entered the interior of the skull close to the left anterior clinoid process; from here it had turned backwards and lodged on the under surface of the brain close to the calcarine fissure. A splinter of bullet had branched off to the left and lodged in the under surface of the hinder part of the temporo-sphenoidal lobe, buried just below the surface. Close to the anterior clinoid process was a small aneurysm of the left internal carotid artery, about three quarters of an inch in diameter, containing *post-mortem* clot. Lodged in its wall was a small splinter of bone. The artery opened directly into the sac, and emerged again above. The sac was smooth-lined, and communicated in front and to its right side with the track of the bullet and

so with the right side of the nose. It was evidently along this track that the fatal hæmorrhage had taken place. The right internal and external carotid arteries were occluded by the ligatures which had been placed upon them. The cortex on the right side appeared to differ in no respect from that on the left. The viscera showed no signs of disease."

Aneurysms of the intra-cranial part of the internal carotid artery are rare. These cases have been included under the term "orbital aneurysm," especially by Rivington, on the grounds that whether the lesion of vessels is in or behind the orbit the symptoms produced are practically similar. Such symptoms, some or all, may be exophthalmos, pulsation of the globe, development of a soft pulsating tumour at the margin of the orbit, especially above, bruit and noises in the head, distended veins and chemosis, and paralysis of the eye muscles and iris. Of all these the prominent symptoms are pulsation of the eyeball and the pulsating tumour at the margin of the globe.

Of the lesions due to trauma which have been included under the term "orbital aneurysm" we have, firstly, the most frequent, an arterio-venous communication between the carotid artery and the cavernous sinus; here a bruit, pulsation, and evidence of venous obstruction should be marked; secondly, only one traumatic case described, where the orbital veins and cranial sinuses were blocked; its diagnosis would be difficult to make, the venous distension as in aneurysmal varix would be present, but on puncturing instead of arterial blood issuing it would be venous; and, thirdly, we have aneurysm of the internal carotid in relation with the cavernous sinus.

The signs of this aneurysm are few. Mr. Pearce Gould suggests the possibility of the ordinary signs of an intra-cranial tumour, as vomiting, headache, and optic neuritis, all of which were absent in our case. Of the symptoms due to pressure on nerves the first evidence noticed was trophic ulceration of the cornea which preceded any anæsthesia. Later sensation was absent over the distribution of the first and second divisions of the fifth nerve, which was quickly succeeded by involvement of the sixth and third nerves. As might be expected where there was no arterio-venous communication, there was no pulsation of the globe. The signs of interference of the return of blood by the ophthalmic veins were very limited, due probably to their free communication with the pterygoid plexus. There was an entire absence of buzzing and other noises in the head.

With regard to the hæmorrhages it is noteworthy that the first was a slight one and occurred on the eleventh day. It may be assumed that there was no actual hole produced in the artery at the time of the injury, and that local softening was caused in the vessel's wall by the contact of the spicule of bone with the addition of sepsis; this hæmorrhage was a true secondary one. Conflicting points in the diagnosis were the lesions of the left eye with the hæmorrhage from the right nostril; the persistence of the bleeding from this side pointed to the right vessel being the one at fault; the course of the bullet track through the ethmoid and sphenoid as shown at the autopsy fully explained why this was. The good effect of ligaturing the right carotid was most striking although on the wrong side; a similar experience has been recorded in other cases.

VI.—*On a Case of Appendix Abscess, followed by Abscess in the Left Iliac Region: subsequent Internal Strangulation; Operation; Recovery.* By STEPHEN PAGET, F.R.C.S. *Read November* 11, 1904.

IT is only what we might expect, that the letting out of an appendix abscess should be followed, in this or that case, by localised suppuration elsewhere in the abdominal cavity; and there is no reason to think that the secondary abscess in such cases is due to the rupture of adhesions shutting off the primary abscess. Often, when we have opened an appendix-abscess, we see a knuckle of small intestine, or a fringe of omentum, moving freely in and out of the cavity, and we wonder how the patient has escaped general peritonitis. Then comes the sudden emptying of the abscess cavity, which alters the conditions of tension and intra-abdominal pressure; the arrangement of the intestines is suddenly displaced, they shift this way and that, accommodating themselves to the new state of things. It is only natural that infection should sometimes be conveyed or sucked into some other region of the abdomen. That is not the fault of the surgeon; he cannot prevent the shifting of the intestines, after the emptying of the abscess cavity, any more than he can prevent them from shifting after the natural emptying of the rectum or the bladder. The wonder is that these secondary abscesses after suppurating appendicitis are not more common.

As an example of this transmission of infection, I would quote the case of a boy, æt. 13, from whose appendix-region I let out five or six ounces of thick fœtid pus. The cavity seemed well shut off, except that the pus had burrowed upward along the outer aspect of the ascending colon, and here there was an inch or two of small intestine moving freely in and out at the edge of the cavity. Nine days later I had to operate on him again for an abscess in the middle line, between the bladder and the rectum, containing nearly a pint. He made a rapid recovery, but he is perhaps in danger of some later trouble from internal strangulation, such as occurred in the case which is the subject of this paper.

Jessie I., æt. 13, an epileptic, was admitted to Hospital

April 22, 1903, with signs of appendix abscess. Operation was done at once. The fluid was sero-purulent, the appendix was sloughing, and on the point of perforation: it was removed, and the cavity was drained with a large tube and with strips of gauze. On May 1, nine days after the operation, there were signs of abscess in the left iliac region; several ounces of pus were let out here. Both the cavities were kept well drained, and healed well.

During the next fourteen months I saw the patient only once or twice. The epileptic fits, which had been in abeyance while her wounds were open, returned as they healed, and became frequent, three or four a week; they occurred mostly at night-time. The bowels, which had given her no trouble before her illness, became constipated, and she had to take medicine for them. From time to time, once every two or three weeks, she had attacks of pain in the lower part of the abdomen. These pains used to occur at night, after she had been in bed and asleep for some hours. They were sometimes very severe, but were relieved by hot fomentations, and lasted only a few hours, and were gone the next morning. She never vomited during them, nor was there any marked distension, but the lower part of the abdomen became rigid and tender, or even so tender that she could not bear to have it touched.

On August 24, 1904, she was taken with severe pain, followed by vomiting and obstruction. On August 26 she was admitted to hospital. On August 29 the abdomen was opened in the middle line, and the intestines were found greatly distended and slightly adherent to the anterior abdominal wall. Some thickened omentum was ligatured and divided; and then, hidden under the distended coils, was found a collapsed coil, deep in the abdominal cavity, tightly nipped under a narrow transverse band no thicker than a 4 or 5 catheter. The division of this band gave immediate relief, and the child made a rapid recovery.

Three points in this case may be worthy of note. First, that the appendix was removed at the first operation. If this had not been done it might have been said that the spread of infection to the opposite iliac fossa, and the subsequent formation of a strangulating band, were due to the non-removal of the appendix.

Next, that the attacks of pain, on and off, for more than a year before the final strangulation, were nocturnal; they occurred all, or nearly all of them, when the child was in bed and had been asleep for some hours. The epileptic fits also

occurred at night; and it may be that the convulsive movement of the trunk-muscles made a loop of intestine slip under the band. But I am more inclined to think that the recumbent posture and the relaxation of the muscles in sleep were among the causes which predisposed the intestine to be strangulated, for I have had two cases of ordinary strangulated hernia occurring in sleep, after the patient had been in bed for some hours.

Finally, it is to be noted that the true significance of these attacks of pain, during a whole year, was overlooked. They were attributed, in a vague general way, to "adhesions." But the existence of a transverse strangulating band might almost have been inferred from the very nature of the case. There had been suppuration in the right iliac region, and it had caused suppuration in the left iliac region. The band was stretched across between them, so that a median incision came straight down on it: which seems to show that, in any case of double suppuration in the abdominal cavity, if the patient at any later period shows signs of intestinal obstruction, the cause of that obstruction must be sought neither at the first centre of suppuration nor at the second, but between them, and will be reached through an incision mid-way between the two scars.

VII.—*The Crises of Posterior Basic Leptomeningitis.* By CHARLES R. BOX, M.D.Lond. *Read November 25, 1904.*

THE non-tuberculous posterior-basic meningitis of infants is a disease which is fairly common and usually recognised with ease. Posterior-basic meningitis occurring in older children and in early adult life—that is to say, after the consolidation of the skull by closure of the fontanelles—may be extremely puzzling in its clinical manifestations.

The question of surgical interference often arises in these cases. Deafness is frequently present and may be unilateral; this, in conjunction with the other symptoms, may suggest either an infective sinus thrombosis or an intra-cranial collection of pus secondary to ear disease. The common occurrence of a purely secondary suppurative otitis media may further complicate the diagnosis. The result is that unnecessary and improper operations are sometimes performed.

Several cases of disease of the kind mentioned have come under my observation of late years, some clinically in the wards of St. Thomas's Hospital, some in the *post-mortem* room of the same institution, and some elsewhere. For permission to record some of the cases on which this paper is based I am indebted to Dr. Hector Mackenzie and to Dr. H. P. Hawkins. I must also express my thanks to Dr. Chappel for permission to use the notes of a most instructive case under his care at Tottenham Hospital. In one of the earlier cases to which my attention was particularly directed I advocated surgical exploration, and by so doing wrought harm rather than good, the operation being conducted on the erroneous assumption that a suppurative process was going on in the brain. This experience led me to investigate other cases which came under my observation for a symptom complex similar to that which had led me astray. I found that its presence was not an isolated occurrence, but on the contrary that it often presented itself clinically.

In its most complete form the complex of symptoms to which I refer, and which I think fairly may be designated "the crisis of posterior basic leptomeningitis" is as follows: The temperature suddenly rises from subnormal or near normal to a considerable height—it may be as much as 7° F.

The patient, who was previously feeling fairly comfortable, experiences a feeling of chillness or even has a rigor. Headache, usually either frontal or vertical, sets in and often is accompanied by vomiting. The neck becomes stiff or retracted, or, if already present, these conditions are exaggerated. The mental condition is generally dull, the patient becoming drowsy or apathetic. The pulse-rate does not fall, but, on the contrary, is usually increased. In the case of one patient the lateral column symptoms, exaggeration of knee-jerks, clonus, etc., were considerably exaggerated during the paroxysms. The temperature remains high for a short time, and then may fall as abruptly as it rose, the urgent symptoms at the same time subsiding. The whole crisis may be over in a few hours, or it may be twelve hours or more before the normal condition is regained. The disturbance mostly is at its maximum in the latter part of the day, usually near midnight.

The temperature charts of patients in whom these crises appear with frequency present a very striking appearance. In some instances they exactly resemble the chart of a quotidian ague, in others that of a tertian fever; and in yet others the occurrence of the paroxysms is more irregular, and they appear at longer intervals or in small groups.

As the disease progresses the febrile paroxysms may entirely disappear, but the case records show that this is not necessarily of good augury, for sudden death may occur, as in Case 2.

It is evident that, given a chart showing the variations I have mentioned and a patient presenting head symptoms, the question of the presence of sinus thrombosis or of one of the suppurative intra-cranial complications of ear disease at once arises. The co-existence of deafness and perhaps of purulent ear discharge serves to render the diagnoiss even more difficult.

The purulent discharge from the ear usually is due to secondary infection from the pharynx *via* the Eustachian tube in a patient more or less stuporous; but in some instances the otitis media appears to be secondary to an otitis interna, which in turn has arisen by an extension of infection along the arachnoid sheath of the auditory nerve.

The deafness may be due to implication of the auditory nerve trunk in its passage from the brain to the internal ear or to implication of the internal ear by extension of infection along the arachnoid sheath of the nerve in the manner above indicated.

The acceleration of pulse during the crises is noteworthy,

contrasting as it does with the retardation which usually occurs in patients suffering from increase of intra-cranial pressure.

Owing to the latency of the nervous symptoms, particularly in the early and intermediate stages of the disease, the patients may come under observation with intermitting or paroxysmal pyrexia of obscure origin. Two of the patients whose histories I report were in the early stages supposed to be suffering from typhoid fever, and a similar suspicion was at one time entertained with regard to a third.

Murchison, in his paper on "Intermitting or Paroxysmal Pyrexia," published in the *Lancet* of May 3, 1879, gives the following summary of the conditions in which such fever may occur:

1. Malarious intermitting fever or true ague.
2. Certain cases of typhoid or enteric fever.
3. Certain cases of relapsing fever.
4. Pyæmia.
5. Fever from pent-up pus, independent of pyæmia.
6. Fever from ulcerative endocarditis and arteritis.
7. Tubercular fever.
8. Fever in lymphadenoma.
9. Syphilitic fever.
10. Urinary intermitting fever.
11. Hepatic intermitting fever.
12. Intermitting fever from the use of morphine.

I have transcribed the list as Murchison published it. To his causes we must add the fever of certain cases of meningitis such as I describe. Both Gowers and Osler made a brief reference to this type of fever, but Bristowe, in a paper communicated to Brain in July, 1888, and reprinted in his volume of *Diseases of the Nervous System* is more explicit. Under the title of "Recovery from Idiopathic Meningitis" he records the case of a porter æt. 19, and states that the temperature was irregular, each access of severe pain being accompanied by a rise. At the end of the paper he mentions remittent febrile attacks, usually accompanied by aggravation of the headache and sickness, among the symptoms which are apt to attend and are fairly distinctive of meningitis at the base of the brain and upper part of the cord.

Case 1.—T. K., male, æt 11. Admitted to St. Thomas's Hospital, August 19, 1901. Died October 30, 1901.

Family history.—Father and mother both alive and healthy. A history of phthisis on the father's side.

Previous history.—Measles four years ago. Has had several attacks of sickness and headache, never lasting more than a day.

Present illness.—Fell on the back of his head two days before admission. Two days later, in the early morning, was seized with severe headache, accompanied by vomiting. The temperature was subsequently found to be 103° F. He was admitted the same day.

State on admission.—Complaint as above. Temperature, 103·4°. Respirations, 20. Slight labial herpes. The lungs, heart, and abdomen showed nothing abnormal. The knee-jerks and plantar reflexes were normal. The urine contained a faint trace of albumen. The serum reaction for typhoid fever was negative.

For the first twelve days the lad was very comfortable in the mornings, but the temperature rose with great regularity on alternate afternoons or evenings from three to five degrees, the maximum being 104·2°. These elevations were usually accompanied by a severe headache, chiefly vertical, and some tenderness at the back of the neck.

During the succeeding week (*the third week of the illness*) the patient was taking quinine mixture (2 grains of sulphate of quinine) three times a day. He was apparently better, and the oscillations of temperature were much less. The temperature was usually raised in the latter part of the day, but rarely rose as high as 101° F. The knee-jerks were present, but were elicited with difficulty. There was no papillitis.

At the commencement of the fourth week the quinine was omitted and *for the next fortnight* the temperature, although subnormal in the mornings, rose with monotonous regularity to a high level in the latter part of the day. The maximum ranged between 102° and 104·6°, being more frequently the latter. The daily elevation coincided with the onset of headache and drowsiness, and sometimes with stiffness of the neck and occasionally vomiting.

During this period the blood was examined (1) for the agglutinative reaction of typhoid fever; (2) for leucocytosis; and (3) by culture for micro-organisms, all with negative result. The patient was now taking a mixture of iodide of potassium and perchloride of mercury. At the end of this fortnight Kernig's sign was obtained for the first time. The knee-jerks were still present, the plantar reflexes of the flexor type and the ocular fundi healthy.

From this time onwards there was less fever, but the

temperature was rarely quite normal. The periodicity of the elevations of temperature was less marked, and the headache was more constant. Early papillitis became evident, and there was occasional incontinence of urine and fæces. Increasing stupor, with loss of knee-jerks and of plantar reflexes, ensued. Emaciation became extreme.

Cultivation of fluid obtained by lumbar puncture yielded either a very small bacillus or a coccus. The growth was of feeble vitality, and died out without giving opportunity for further investigation. No tubercle bacilli were found in the fluid. Nasal feeding ultimately became necessary, and the patient died after an illness of seventy-two days' duration.

Successful lumbar puncture was performed on three occasions—October 22, yielding 13 drachms; October 23, 23 drachms; and October 30, 42 drachms.

Post-mortem.—The body was greatly emaciated. The ventricular system of the brain was much distended and the convolutions were flattened by pressure. Inflammatory exudation was present at the base of the brain on the medulla, pons, and interpeduncular space. Numerous small ecchymoses were found on the cerebral dura mater, possibly as the result of the lumbar punctures. The lungs were œdematous. No signs of tuberculosis were found anywhere.

Duration, seventy-two days.

Case 2: Tottenham Hospital, Dr. Chappel's case.—A. H., female, æt 10. Came under observation January 31, 1903. Died, May 24, same year. The patient was admitted with a history of headache and vomiting. Duration not stated.

On examination.—Body and limbs much emaciated. Nothing abnormal detected in the thoracic or abdominal viscera. No glandular enlargements. Urine normal. Temperature, 102° F. Respirations, 28. Pulse, 28.

For the first week in hospital the temperature rose daily—sometimes in the morning, sometimes later—to 102° or 103° F. In the intervals it approached normal, or was even subnormal. One of these elevations was attended by an attack of vomiting and headache. The pupils were noted as "not dilated."

During the second week the general outline of the chart was similar, but the maximal temperatures gradually diminished from 103·7° to 99·8°. The pulse rate varied; it was not slowed, and acceleration corresponded to the elevations of temperature. On one occasion during this week some rigidity of the neck was noticed; its onset coincided with an elevation

of temperature, and, at the same time, the pupils were noted as "unequal."

During the third, fourth, fifth, sixth, seventh, and eighth weeks the temperature curve was of a striking character, being subnormal in the early part of the day and rising, usually to 103° or 104° F., later on. The pulse quickened when the temperature rose, but the respirations did not increase in a proportionate degree. Severe headache, and sometimes vomiting, accompanied the febrile exacerbations. The patient usually lay curled up in bed in a drowsy condition. There was no indication of otitis media, and there was no optic neuritis. Emaciation was extreme, and the belly was retracted. During the fourth, fifth, and sixth weeks sulphate of quinine was administered daily at 3 p.m. in three-grain doses. This possibly had some effect in staving off the crises when the time of administration corresponded to the lull before the elevation of temperature, but of this it is not possible to speak positively.

With some remission of the fever in the ninth and tenth weeks the patient's condition appeared to improve, her headaches became less frequent, and she was finally allowed to get up. She had been carefully examined for indications of intra-cranial suppuration, but no definite indications were forthcoming. She was now taking a mixture containing 7 grains of salicylate of soda and 4 grains of iodide of potassium every six hours.

At the commencement of the eleventh week of observation she had, without warning, a fit. She cried out, fell down unconscious, and became rigid all over. There was no passage of urine or of fæces during the seizure. She was put to bed, and for the three succeeding days was said to be delirious. Finally, she became unable to speak or to swallow. These urgent symptoms, however, proved transitory. Fits again recurred at intervals accompanied by much vomiting. In some of these fits clonic spasm was noticed, followed by rigidity and opisthotonos. There was still no optic neuritis.

Finally she lost control of the sphincters and died. For the last three weeks the temperature was mostly at subnormal levels and never febrile.

She was under observation for 113 days.

The necropsy revealed distension of the cerebral ventricles and evidence of some posterior-basic meningitis.

Duration, practically four months.

Case 3.—John U., æt. 10. Admitted to St. Thomas's

Hospital, January 23, 1903. Died July 1, same year. Bronchitis every winter for the past five years. In December, 1902, he had an attack of varicella, from which he was recovering at the time of the fall to which his present illness was attributed.

Present illness.—On December 15, 1902, he fell down a flight of sixteen stone steps. He complained of his legs, but no abrasions or bruises were discovered. The same day, after the fall, he complained of pains in his head, and vomited. There was no shivering. For the next nine days he was under treatment at home with an ice-bag applied to his head. The headache continued, there was occasional vomiting, and the bowels were confined. No stiffness of the neck was recognised.

At the end of this period he was removed to the Park Hospital, at Catford, and there his condition was noted as follows:

Admitted with headache; retraction of head; well-marked Kernig's sign, and a temperature of 102° F. For past ten days has had headache, head retraction, and increased sensitiveness to light and sound. The serum reaction for typhoid fever proved negative, and the optic discs were found to be normal in appearance.

On every third day, from January 3 onwards, the temperature rose to 102° to 103° with vomiting. The periods of pyrexia lasted for from twelve to eighteen hours. In the intervals the patient was free from discomfort.

The boy was admitted to St. Thomas's Hospital, under the care of Dr. H. P. Hawkins, on January 3, 1904. He was greatly emaciated and very irritable. He complained of tenderness of the back of the head, but showed no retraction of the neck. There was no cranial nerve paralysis, neither were there any alterations in the pupils or the optic discs. The hands and tongue were tremulous; knee-jerks normal; plantar reflexes not obtained; Kernig's sign present. The lungs, heart, and abdominal viscera were normal. The urine contained no albumen or sugar. Pulse 96°, regular; respirations normal.

For the first week the temperature remained normal or sub-normal in the mornings, but rose in the afternoons or evenings, with onset of frontal headache, vomiting, and irritability. On the second day of observation the temperature reached a maximum of 103°, but after that the degree of pyrexia was less, the temperature during the exacerbations only reaching 89° at the end of the week. No further alteration in the physical signs.

During the second week the lad was very comfortable, and there was practically no fever.

The condition for the succeeding eleven or twelve weeks was as follows: Short febrile periods lasting from three to five days recurred. The chart was spiked, the evening or afternoon elevation increasing for one or two days to a maximum, and then gradually falling again. The morning temperatures were mostly either normal or slightly febrile. Headache and vomiting often, but not always, corresponded with the elevations. Apyretic intervals of from one to four days intervened between the attacks, and during these the patient was free from all discomfort.

Towards the end of this period exaggeration of the knee-jerks, and a tendency to ankle clonus appeared, both of which signs were better marked during the febrile attacks than in the intervals. Rarely the Babinski sign was present, and there was also some doubtful nystagmus. Tremors of lips and tongue, and unsteadiness of gait were very marked. Signs of bronchitis appeared at the end of this time.

At the end of this period, and for the succeeding seven weeks, the febrile paroxysms were wanting, the temperature being only very occasionally elevated. The headache was now less, the exaggeration of reflexes not so marked, and vomiting was only occasional. Emaciation was progressive, and towards the end of the period the boy began to refuse his food, necessitating nasal feeding on one occasion. He began to complain of pain all over his body, and had one or two screaming and emotional attacks. The temperature now became sub-normal, and there was some indication of loss of control over the rectum. He lapsed into unconsciousness at times. On July 1 he had a rigor, during which the pulse exceeded 200 per minute, and the respirations were 158. Finally, he died somewhat suddenly with intense dyspnœa. The duration of illness was 198 days.

The treatment throughout was symptomatic—opium and various analgesic and antipyretic drugs, occasional rectal and nasal feeding. Duration of illness over six months.

Post mortem.—The body was very emaciated. The dura mater, the great sinuses, and the bones of the skull were quite healthy. The cerebral convolutions were flattened and the surface was sticky. During removal of the brain clear serum, evidently under great pressure, escaped from the transverse fissure and its choroidal prolongations. Signs of a past lepto-meningitis in the form of opacity and patchy thickening could be traced from the region of the lamina

cinerea in front, as far as the cerebellum behind. The change was also traced on to the upper surface of the cerebellum in its anterior part. The lips of the Sylvian fissures were adherent. No tubercles were seen. There was considerable distension of all the ventricles. The fluid in them was clear; the walls of the ventricles were not appreciably thickened, and the sutures of the skull had not yielded. The substance of the hemispheres, the pons, and cerebellum appeared normal. The pia-arachnoid of the cord was œdematous, and showed signs of past inflammation on its dorsal aspect along its whole length. There was a disseminated broncho-pneumonia which had not broken down. No trace of past typhoid fever in the intestines. A culture from the meninges at the time of the *post-mortem* examination yielded a growth of streptococci.

Case 4.—H. K., æt. 9, admitted to St. Thomas's Hospital on February 8, 1901; died March 19, 1901.

Family history.—Father and mother quite healthy. An elder brother of patient said to be suffering from pulmonary tuberculosis.

Previous health.—Had discharge from the left ear in infancy but none since. Measles when 3 years old. Occasionally suffers from headaches, which usually come on at night.

Present illness.—Fourteen days before admission the lad awoke in the morning with pain in the left temporal region, and shortly afterwards he vomited. There had been no premonitory symptoms, and there was no history of injury to the head. The headache persisted, becoming frontal, but the vomiting ceased after five days. There were no fits.

On admission.—He appeared very drowsy and was slow in answering questions, but when roused up appeared perfectly rational. There was slight stiffness of the neck and some tendency to spasm in the hamstrings. Muscular weakness was general but no definite paralysis was detected. The knee-jerks were brisk, and the plantar reflexes were indeterminate. The pupils were equal and active, the ocular fundi normal. There was no discharge from ears or from nose. The frontal and maxillary sinuses were examined by transillumination and no evidence of pus found. The lungs, heart, abdomen, and urine were normal. The respirations were irregular. Temperature 102·6°. A blood examination on the day after admission showed a slight increase in the polymorphonuclear cells.

For the first week after admission there was continuous

slight fever, with a daily maximum of 102°. The headache persisted. Although the left membrana tympani appeared healthy, the left ear was completely deaf, even on contact. The serum test for typhoid fever proved negative, and lumbar puncture in two places failed to draw any cerebro-spinal fluid.

During the second week of observation the temperature oscillated more, being at times nearly normal, and at some other time of the day rising to about 102° F. The pulse was always accelerated, varying between 90 and 104. The patient continued to complain of frontal headache and was very intolerant of interference of any sort. The pupils were large and their reactions sluggish. There was still no papillitis. Neither knee-jerk could now be obtained, and the big toe on the left side was fairly constantly retracted on stimulation of the sole of the foot. The boy remained absolutely deaf in the left ear and emaciation was very rapid. *At the end of this period the left lobe of the cerebellum and the left temporo-sphenoidal lobe were explored with a negative result.*

During the third week of observation the temperature was normal and the pulse 108 or thereabouts. The headache was much better and the left knee-jerk reappeared. The exploration wounds did well.

In the fifth week there was a re-accession of fever. A rigor, with temperature of 103°, vomiting, and severe headache occurred. At the same time there was some abdominal pain. As there was nothing in the wounds to account for the condition, *the left mastoid and also the left frontal lobe were explored;* again the result was quite negative. The temperature remained about 102° for the next two days. Each night the patient was very restless, crying out and occasionally vomiting; in the mornings he was drowsy and inclined to sleep. As night came on he again became noisy and restless, shouting and complaining of his head. As a rule the quiet and drowsy periods corresponded to remissions of fever.

Signs of broncho-pneumonia appeared towards the end. There was still no papillitis. Both knee-jerks were again absent, and the plantar reflexes were variable. The pulse was never abnormally slow. The duration of illness was fifty-three days.

Post mortem.—Extreme emaciation. Definite post-basic meningitis, with yellowish-green exudation, extending as far forwards as the optic chiasma. Cerebral ventricles greatly distended with slightly turbid fluid and their walls softened. No excess of fluid in the sheath of the spinal cord, but a few patches of chronic inflammation were present on the anterior

surface of the spinal arachnoid, and a small meningeal hæmorrhage was found at the site of the lumbar punctures. The ears and the air-sinuses were healthy. The deafness was probably due to involvement of the auditory nerve at the base of the brain. The lungs showed extensive bronchopneumonia. No tuberculosis.

Case 5.—J. B., æt. 37. Previous history unknown.

On March 24, 1900, he was seized with shivering and a feeling of malaise. He was found to be feverish, and on April 3 complained of abdominal pain. He was supposed to be suffering from typhoid fever. On April 4 his mental state was peculiar; he was obstinate, shivering and collapsed, but not delirious. No paralysis was detected. He was removed to hospital on this day.

On April 5 a slight discharge from the right ear was noticed for the first time. The temperature in the morning was 101·4° F. He appeared stupid, but actively resisted all attention and examination. The pulse was 96, and there was no sickness or cranial nerve paralysis. A systolic murmur was present at the impulse of the heart, but the lungs and abdominal viscera appeared normal. A slight convulsive seizure occurred on this date. On April 8 he was rambling and incoherent. He was thought to be deaf. The urine contained no albumen.

On April 10, 12, and 14 he had attacks of frontal headache, with rigors. These attacks corresponded with elevations of the temperature, which on the last occasion reached 102·3°, and was accompanied by slight twitching of the right side. The appearance of the optic discs gave rise to a suspicion of commencing optic neuritis (temporal side) on April 16. He now began to complain of pain in the right cheek, which shortly afterwards extended also to the lower jaw of this side. Frontal headaches still accompanied the accessions of fever, but the maxima of temperature were less. He frequently vomited. The pulse as a rule became accelerated when the temperature rose.

At the end of April he began to pass his evacuations under him and was more stuporous. The tendon reflexes were exaggerated and the body rigid. Headache and vomiting still occurred occasionally.

Early in May definite head retraction was first noticed. There was now definite papillitis in the right eye, and slighter but distinct papillitis in the left.

On May 7 difficulty in swallowing appeared, and coma and death supervened.

Duration of illness, forty-four days.

The *post-mortem* examination showed only a basal meningitis with considerable distension of the cerebral ventricles.

Concluding Remarks.

In the first three cases read the crises were present at some stage or other of the illness and were of typical character.

In the other two cases, although exacerbations corresponding to crises were present, they occurred in a modified form and were not so definite or so striking as in the first three patients.

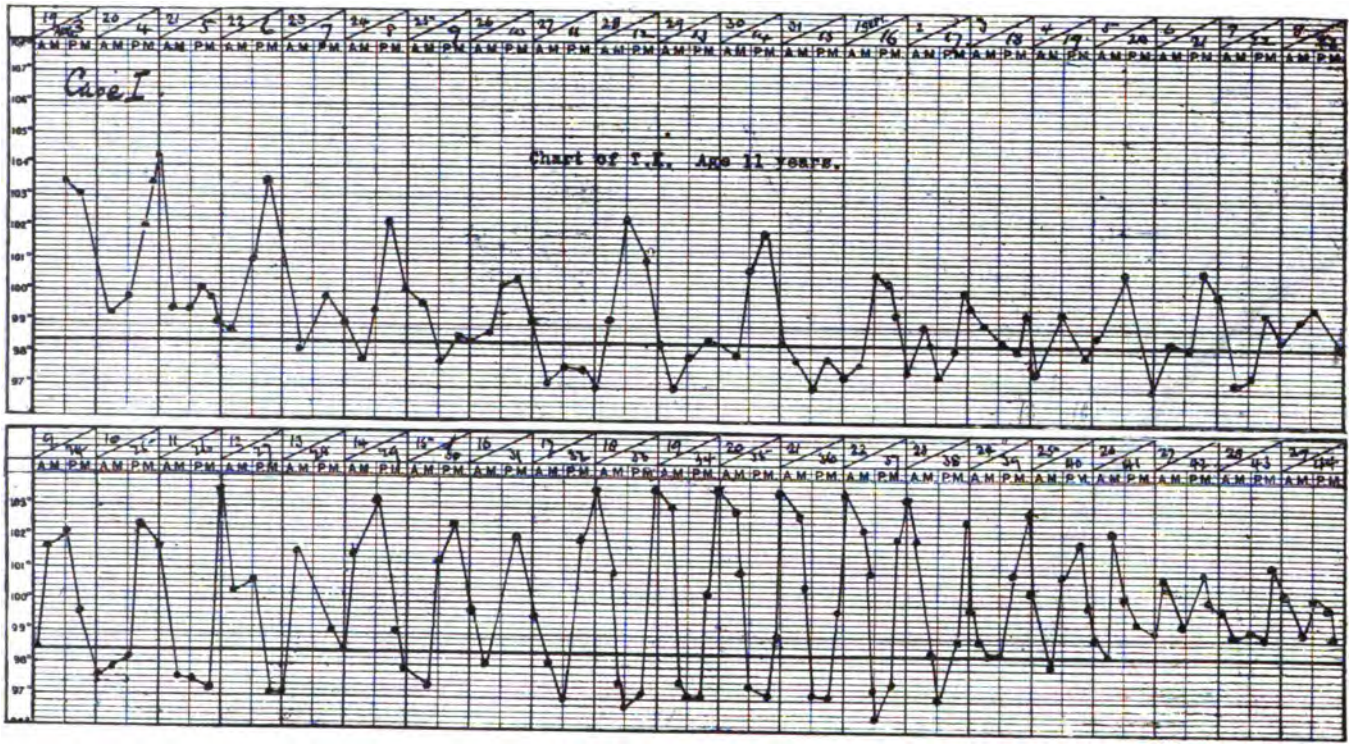
I am at a loss for a definite explanation of these curious exacerbations of symptoms; but think they are due to temporary increase in the pressure in the ventricular system of the brain, for most of the cases finally show some degree of hydrocephalus *post mortem* and in all the cases I have recorded the fontanelles were closed, the age of the youngest patient being 9 years and that of the oldest 37 years.

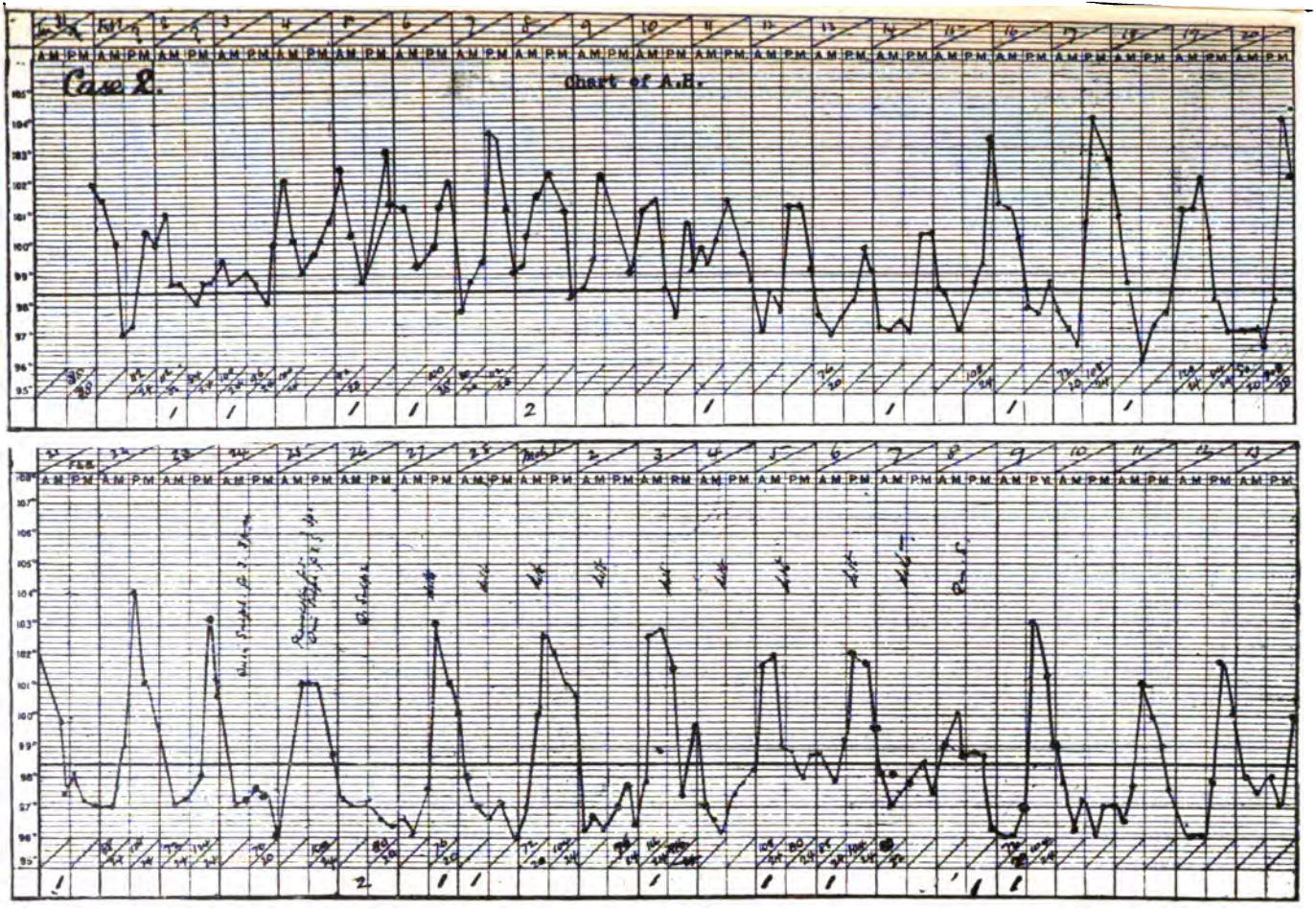
There still remains, however, a difficulty in accounting for the pulse rate during the crises. The pulse is accelerated instead of retarded, as we should expect from an increase of intra-cranial pressure. A further enigma is the periodicity with which the crises recur.

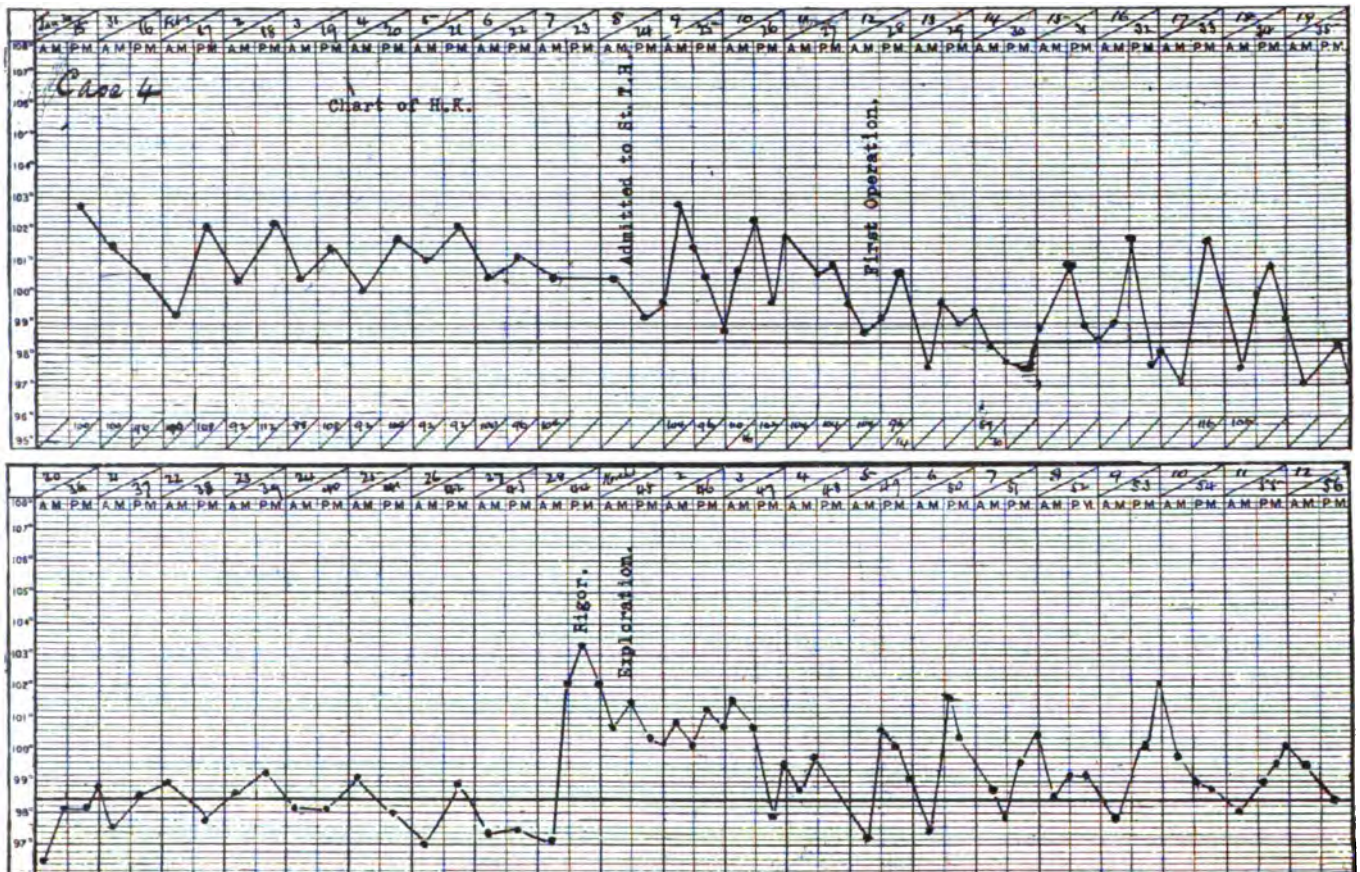
It is just possible that a more extended knowledge of the bacteriology of the disease may shed light on the cause of the exacerbations, for we cannot help recalling the similar variations which occur in malarial infections.

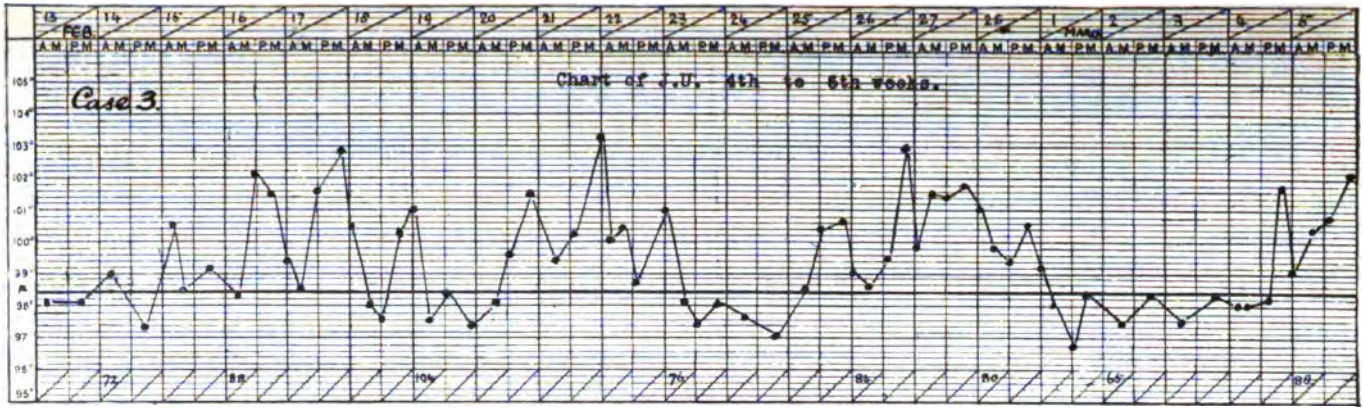
If a periodic increase in the intra-ventricular pressure is the correct explanation of the crises, they should be relieved by lumbar puncture; for despite the current teaching that the foramina in the fourth ventricle are occluded in these cases I quite agree with those who state that the communication often persists. The freedom of the communication I have many times demonstrated in the *post-mortem* room by draining the ventricles through the spinal theca before removing the skull cap. The question also arises whether drainage of a more permanent character than lumbar puncture affords is to be recommended in cases such as I have recorded.

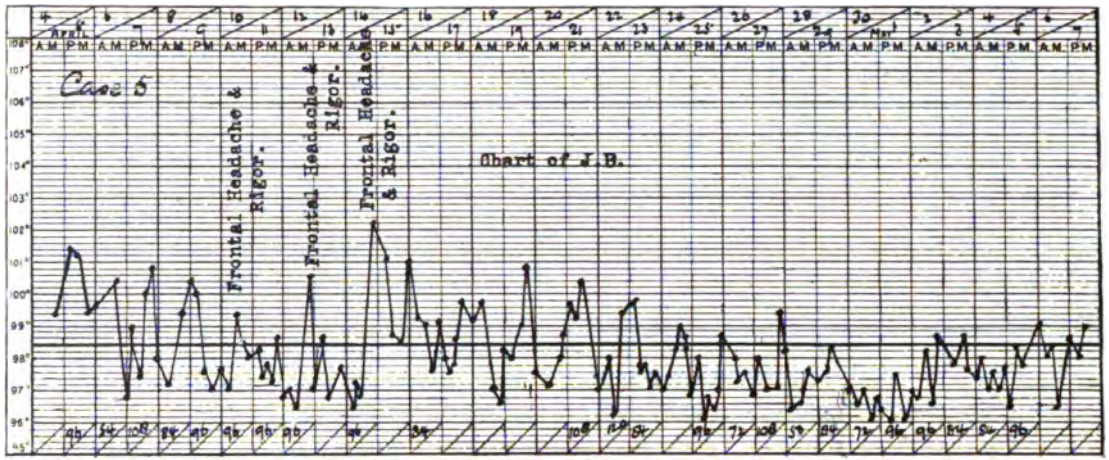
In several cases quinine seemed to have some influence in mitigating the exacerbations.


Case I.
Chart of T.E. Age 11 years.


Case 2.
Chart of A.B.r.


Case 4
Chart of H.K.
Admitted to St. T.H.
First Operation.
Rigor.
Explanation.


Case 3.
Chart of J.U. 4th to 6th weeks.
FEB.
MARCH


Case 5
Chart of J.B.
Frontal Headache & Rigor.
Frontal Headache & Rigor.
Frontal Headache & Rigor.
April
May

VIII.—*A Case of Unilateral Tuberculous Meningitis in which a Bullous Eruption followed Lumbar Puncture.*

By H. D. Rolleston, M.D., and B. N. Tebbs, M.D.

Read November 25, 1904.

A BOY, æt. 5, was admitted to St. George's Hospital at 11.30 p.m. on March 3, 1904, suffering from general convulsions. There had been practically no symptoms of illness till the day of admission, when he complained of slight malaise, and vomited once. He was suddenly seized with general convulsions at 8 p.m., the mouth, at the onset, being said to have been drawn to the left. On admission the child was deeply unconscious, the temperature was a little over 107°, the pulse 200, and respiration of the Cheyne-Stokes type. There was involuntary passage of urine and fæces. The pupils were widely dilated, equal and not re-acting to light; the corneæ were insensitive. General twitching of the face, trunk and extremities was present, together with a considerable degree of general tonic contraction. The head was forcibly turned to the right, and there was conjugate deviation of the eyes in the same direction. There was no opisthotonos, but the body was arched as a whole to the right, the left side being more flaccid generally than the other. The symptoms seemed to point to some irritative lesion of the left motor area, and a tentative diagnosis of meningitis, affecting chiefly the cortical areas, was made. Nothing abnormal was detected in the fundi on ophthalmoscopic examination. The heart appeared to be quite normal. The convulsions ceased after the patient had been put to bed, and the temperature was brought down by cold sponging. A considerable amount of general tonic contraction, however, persisted, and this was found to become so much more marked on the application of any stimulus that the cold sponging had to be abandoned, and an ice-bag to the head was substituted. The patient remained in a state of coma from the time of admission up to death, on March 7. On the day following admission the child was quite comatose, lying with the limbs extended, in a state of tonic contraction, twitching occurring only on the application of stimuli. The right side of the body was more rigid than the left. The extremities were kept in a state of rigid extension,

with the exception of the hands, which were markedly flexed, and the feet, which were in a condition of plantar flexion. The abdomen was slightly retracted. There was no retraction of the head. The knee-jerks were present, the left being brisker than the right, but no clonus could be obtained. Kernig's sign was absent. The right pupil was larger than the left, and both reacted very slightly to light. Two small urticarial wheals were present, one over the right elbow and another over the back of the right wrist. Lumbar puncture was performed on this day; the needle was entered between the spines of the third and fourth lumbar vertebræ, and was left in for six hours, with the result that a considerable amount of fluid drained away. The fluid was perfectly clear, and was sterile on cultivation, pointing to a probable tuberculous origin. The relief of pressure due to the withdrawal of the fluid was followed by slight amelioration of the symptoms—the child moaning when disturbed; but consciousness was never regained. On March 6 a Southey's cannula was introduced into the spinal theca between the third and fourth lumbar spines, with a view to relieving pressure. About four ounces of fluid came away altogether, the first portion of it being blood-stained. On the morning of March 7 two bullæ were found to have developed on the outer aspect of the little toe of the right side. One, about a quarter of an inch in diameter, was situated over the terminal phalanx; and the other, half an inch in diameter, over the metatarso-phalangeal joint. The appearance was absolutely spontaneous (hot bottles had not been applied). Death occurred at 4 p.m. on the afternoon of the 7th; the temperature, which at the time of death was 103·4°, subsequently rose to 107°.

At the autopsy, performed by Dr. Trevor, on March 8, there was general engorgement of the intra-cranial sinuses and of the pial vessels. The brain weighed 3 lbs., its convolutions were flattened, but there was no distension of the ventricles. The left Sylvian fissure was matted with lymph, and the branches of the left middle cerebral artery studded with tubercles. These could be traced on the vessels right up to the vertex, and could easily be made out on the vessels dipping down from the pia into the cortex. There was a well-marked collection of tubercles on the second left frontal convolution, over the area, stimulation of which causes conjugate deviation of the eyes to the opposite side. There were no tubercles visible to the naked eye elsewhere in the brain, but one was found on the inner surface of the spinal

dura. In the lower part of the spinal theca, under the arachnoid, there was an elongated clot about 3 inches long, apparently due to hæmorrhage caused by the lumbar puncture. This was lying on the right side, over the posterior roots of the nerves going to form the cauda equina. It is conceivable that the spontaneous appearance of bullæ on the right foot, coming on shortly after the lumbar puncture was performed, may have been associated with irritation of the posterior roots by this clot. The primary tuberculous focus was found in some caseating bronchial glands. The apex of the lower lobe of the left lung was puckered, and on section was shown to contain a cavity the size of a cherry which was in direct connection with a caseating infra-tracheal gland. There was no miliary tuberculosis of the lungs, but only a slight bronchitis, signs of which were evident during life. Some of the mesenteric glands were tuberculous, but with this exception and a certain amount of perisplenitis, the abdominal viscera were healthy and perfectly free from tubercle.

Remarks.

The points of interest about this case are :

(1) The unilateral tuberculosis of the cerebral meninges and the extremely limited area of tuberculous infection in the body, the old lesions being in the mesenteric and infra-tracheal glands, and the miliary tuberculosis in the left Sylvian fissure of the brain and a single tubercle on the theca of the spinal cord. It would appear probable that a mass of tuberculous material must have discharged into a branch of the pulmonary veins and passed almost intact, as an embolus, into the left middle cerebral artery, where it disintegrated or broke up like a shrapnel shell and induced localised miliary tuberculosis.

(2) The association of a hæmorrhage inside the theca vertebralis in contact with the posterior roots and the development of a bullous eruption on the corresponding foot. Can the hæmorrhage which followed, and was almost undoubtedly due to, lumbar puncture, have irritated the posterior roots and produced the bullous eruption ?

On the other hand, were the bullæ entirely independent of lumbar puncture and of the same nature as the two small urticarial wheals which appeared over the right elbow and right wrist before lumbar puncture was performed ?

IX.—*Post-operative Acute Local Tuberculous Infection.*

By EDRED M. CORNER AND LEONARD S. DUDGEON.

Read November 25, 1904.

WE bring this communication before the Society because we wish to draw attention to an important pathological process, local tuberculous infection, and at the same time suggest an explanation for many common clinical manifestations which have never been fully understood. We also wish to cite a few instances which have occurred in our experience, and which illustrate the occurrence of local tuberculous infection. By these few examples, we hope to indicate how widespread and frequent this infection may be, and how little the true significance of the swelling which may appear after operative interference on a tuberculous lesion is appreciated. The occurrence and persistence of the swelling is an indication for further operative measures to be undertaken, such as incision, drainage, and erasion. There is a minor point which deserves especial mention from the clinical point of view, namely, that all the cases of acute local tuberculous infection have occurred when the wound has been closed and has healed by first intention, and not when drainage has been employed or suppuration has taken place.

Case of tuberculous glands of neck; operation; acute local infection of the wound area with tuberculous disease; further operation; recovery.—P. P., a little boy æt. 4, was seen early in October, 1903. He had suffered from a swelling in the right submaxillary region for some time. On October the 9th, the gland was excised and found to be tuberculous and beginning to caseate. Above it was found a second and smaller tuberculous gland, which on account of its adhesions could not be completely excised. The portion which remained was thoroughly curetted. The various structures which were seen in the wound were perfectly healthy—for instance, the sternomastoid muscle. The skin was sewn up completely, and the wound healed by first intention. On the tenth day, it was noticed that there was considerable swelling in the region of the wound, and a director was inserted to ascertain

if there was a collection of fluid present. An insignificant amount of blood-stained serum was found. On November 25th, as the swelling in the neck had rather increased than diminished in the seven weeks which had elapsed, a further operation was advised, and consented to. A few hard masses could be felt round the margin of the old incision, and proved to be diseased lymphatic glands. It was found at the operation on the 28th day of the same month, that all the structures which were seen to be healthy at the first operation, such as the sternomastoid, were now diseased. The involved structures, where it was possible, were excised. This procedure involved a prolonged dissection, exposing all the great vessels and nerves on that side of the neck. The wound, which was drained, was only closed partially. The course of convalescence was quite uneventful. At the present time, the boy is very well and shows remarkably little evidence of the loss of the upper half of his right sternomastoid.

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The portions removed consisted of some lymphatic glands, the upper half of the right sternomastoid, and other tissues of the neck.

Pathological Report.

Lymphatic glands.—The glands show well-marked caseation and overgrowth of fibrous tissue. The lymphoid corpuscles are almost entirely replaced by endothelial and multinucleated cells. Giant-cell formation is extremely well shown throughout the various sections of the glands, especially in and around the areas of caseation. The giant cells are represented chiefly by the large type with many nuclei, which are most numerous around the periphery of the cell. Giant-cell systems can be seen scattered throughout the various sections, and in one of the microscopical specimens, a typical tubercle can be distinguished in the wall of a small blood-vessel. A few tubercle bacilli were found after a careful examination of many microscopical sections.

Tissues from the neck.—Small pieces from the muscular tissues were placed in various fixing fluids. Paraffin sections were cut and stained by Van Gieson's method, hæmalum and eosin, and for tubercle bacilli, but no tubercle bacilli were found in any of the sections.

The muscle-fibres show all varieties of degeneration. Some of the bundles are only slightly affected, but others are completely degenerated. By means of Van Gieson's stain, we are

able to study the changes in the muscle-fibres most accurately. We see that some are stained reddish-brown, others lemon-yellow, orange, bright red, and lastly some are bright red at one part of the cell and lemon yellow at the other. Most of the fibres which are stained bright red or lemon-yellow do not show any cross striation, only longitudinal striation, while others have a homogeneous appearance. Many of the muscle cells are vacuolated and deeply fissured. Multiplication of the nuclei of the muscle sheaths is most characteristic. Large numbers of giant cells are found to be scattered throughout the muscular tissue, and there is also well-marked round-celled infiltration. Caseation has occurred throughout the tissues of the neck.

This case is an instance of an acute infection of the operation area with tubercle. Such cases have previously been misunderstood and deemed to be merely inflammatory. We shall give a few examples, though many more could be cited, in order to illustrate our contention.

Case of tuberculous glands of neck, operation followed by swelling, incision, recovery.—A boy, æt. 6, had enlarged glands in both submaxillary regions, and on each side of the neck they were beginning to break down. First one side of the neck and then the other was operated upon, a week being allowed to elapse in between. After a few days or a fortnight, both sides were found to be swollen, and soon were as large as they had ever been. The wounds had healed by first intention. All concerned in the case were naturally disappointed at this result. The sites of the previous operation were thoroughly opened up, and were found to be full of greyish granulations, which were scraped and left wide open to heal slowly. This case occurred in 1902, before the one related above, but its interpretation is now obvious as an example of local tuberculous infection. The boy is now in excellent health.

Case of recurrent tuberculous glands : repeated operations.—A man, æt. about 25, had had a large number of operations for tuberculous glands of neck. His neck was much thickened with the formation of a large amount of subcutaneous and deeper scar tissue. Each operation was followed by some local swelling, which in the course of time became smaller and harder, like the rest of his neck. Interpreting this case in the light of the first which we have brought before the Society to-night, there can be little doubt that the post-operative

swellings were due to *local infection with the tubercle bacillus*. And the case illustrates another point, namely, that these infections, as would be expected, may quiet down and heal, becoming nothing but a mass of scar-tissue. Hence, it would seem that a large amount of fibrous tissue in the area of operation, so often seen in cases which have undergone many operations, indicates that there has been a slow consolidation and healing of tuberculous tissue. There is certainly more scar-tissue in these regions than accompanies the ordinary healing processes such as is seen in other wounds. The occurrence and healing of tuberculous tissue will explain it.

Case of tuberculous glands; excision; healing by first intention; subsequent breaking down of the scar.—A girl, æt. 9, had some glands excised from her neck, the wound healing by first intention. She was sent with a collodion dressing to the Convalescent Home at Highgate, belonging to the Children's Hospital, Great Ormond Street. After a week's residence there, it was found that the whole length of the scar had opened of its own accord, and there was a broad granulating area exposed, which healed slowly, the girl in the end being discharged quite well. This seems to be an example of a case in which the tuberculous infection did not heal, as in that just related, but which broke down and discharged.

The above examples should suffice for the subject of tuberculous infection of glands, and illustrate its occurrence as a swelling, as the cause of delay in healing, and the formation of an ulcer. Two more cases will be given to illustrate the process in other regions.

Tuberculous disease of knee; arthrectomy; local infection with tubercle; further operation; recovery.—L. T., æt. 4, had a tuberculous knee, for which an arthrectomy was done. The wound healed, but the knee soon began to swell, and later, "tuberculous" granulations appeared in two places in the scar. Treatment was pursued in the hope that the surrounding inflammation would lead to the fibrosis and healing of the infected tissue, but this did not occur. In two and a half months time, it became necessary to completely open up the joint, which was left so. The wound then healed quickly, and the subsequent flexion was corrected later by an apparatus.

Just as we have stated as regards cases of tuberculous glands of neck, so instances of thickening following upon

arthrectomies of joints are of common occurrence. Also, the scars after these operations have been frequently observed to stretch, and then give way, leaving broad ulcerating surfaces. It would be wearying to continue the recital of examples about the knee, hip, elbow, and other joints and regions; and only one more instance will be related, which followed upon the excision of a tuberculous kidney.

Case of tuberculous kidney; excision; infection of the wound with the urine.—K. M., æt. 25, suffered from a tuberculous kidney, which was excised. At the operation care was taken to prevent the soiling of the wound with tuberculous material, but it was noticed that in passing a probe down the ureter a good deal of urine escaped into the depths of the wound. No particular notice was taken of it at the time, and only subsequent events redirected attention to the fact. The wound was completely sewn up and healed by first intention. At the end of a fortnight, it was noticed that the temperature every now and then rose a little at night. A careful examination revealed some tenderness and indefinite thickening in the loin. At the end of the sixth week after operation, a sinus formed. Subsequently, the swelling of the tissues became much smaller and the sinus much shallower, all the signs of healing being present. This is the state of the patient now, and it cannot be said whether or not closure will take place without erasion.*

In this case the infection may have been caused by other organisms than tubercle bacilli, but tubercle bacilli were present in the urine at this time.

It is of the greatest interest to note that, in most cases, the local infection has occurred in wounds which had healed by first intention. This opens up the whole subject of the draining of such operation wounds. Drainage frequently will convert the tuberculous lesion into a septic one. Doubtless, many tubercular cases would be much better if the wounds were incompletely sutured and drainage was employed, but we must be guided by the situation of such wounds. For instance, it is much better to have a tuberculous infection of the knee-joint than one due to pyogenic organisms. The same remark applies to the peritoneum and many other situations where the primary disease may be. In regions like the loin, it is far better to employ drainage.

Finally, a word must be said as to the course to be

* By the time this proof was corrected the sinus had practically healed.

followed in the treatment after the diagnosis of tuberculous infection has been made. This, of course, must be decided by the situation of the disease. In the first place, a sufficient period of time must be allowed to elapse to see if the infection is going to subside and heal of itself. If this happy result is not going to occur, it is necessary either to repeat the former operative procedures with or without the employment of drainage. Ideally, the latter treatment consists of incision, erasion, and leaving the wound open, a procedure which may be regarded as an example of the open-air method. It is not, of course, necessary always to leave the whole wound open.

At the first operation, it is advisable to drain wherever there seems a possibility of the occurrence of tuberculous infection, rather than to completely close the wound in order to reap the more showy result of union by first intention. The one is the sounder course to pursue; the other is the more brilliant, but the less reliable.

X.—*A Series of Intussusceptions in Children.* By Cuthbert Wallace. *Read December* 9, 1904.

THIS series is made up of 20 cases; 12 were males and 8 were females. They were treated at St. Thomas's Hospital and at the East London Hospital for Children, between the years 1898 and 1904. The cases were consecutive and include all that have passed through my hands. The results are good, I think, but they only represent the improvement that has taken place in all hospitals of late years, due to the now almost universal custom of speedy operative interference, which is largely due to the efforts of Mr. A. E. Barker.

Nature of the intussusception.—Of the 20 cases, 19 were entero-colic—that is, involved both the large and small guts; the remaining case was of the colic variety. Of the former class 10 were single and 9 were double intussusceptions. The single intussusceptions were of the ordinary ileocæcal type. It is, however, possible that in two the starting-point was an invagination of the outer wall of the cæcum, since after the reduction of the tumour this part of the cæcal wall remained invaginated, and was with difficulty made to resume its normal shape. Of the double tumours there were four cases of the enteric-ileocæcal (iliaca-ileo-colica) and five cases of the ileocolic-ileocæcal (ileocolic-colic) varieties.

These facts can be tabulated as follows:

Number of cases	20
Small and large gut involved	19
Large gut involved	1

(1)	Ileocæcal	10, or 50 per cent.
(2)	Ileocolic-ileocæcal (ileocolic-colic)	5, or 25 per cent.
(3)	Enteric-ileocæcal (iliaca-ileo-colica)	4, or 20 per cent.
(4)	Colic	1, or 5 per cent.
		20, or 100 per cent.

The diagnosis of the enteric-ileocæcal was made if an enteric intussusception remained after the large gut had been entirely reduced. An ileocolic-ileocæcal (ileocolic-colic) intussusception was diagnosed if, after the reappearance of the cæcum and the appendix, there was still small intestine prolapsed through the valve.

On account of the frequent occurrence of double intussusceptions Mr. E. M. Corner, in a very interesting paper,* has lately expressed his opinion that the accepted ideas as to the comparative frequency of the different forms of intussusception must undergo a modification. Mr. Corner has prepared a table showing what he believes to be the relative frequency of the different varieties, and has proposed a nomenclature for the double intussusceptions.

My own series certainly bear out the contention that double tumours are more frequent than has hitherto been considered to be the case. It is, however, open to doubt that a useful purpose is served by a complicated classification.

If we take the case of those intussusceptions that originate near the ileocæcal valve, we find that they are all of the ileocæcal type, and that the main characteristics of the intussusceptions are identical as regards their appearance and the structures forming their walls; and it is only after the reduction of the main tumour that the presence of a second intussusception allows a differential diagnosis to be made. The ileocæcal region of the intestinal tract is a complicated one, and having attracted much attention, is more richly endowed with names than any other region of the same length.

With a little ingenuity a very extensive nomenclature can be made out for intussusceptions arising in the neighbourhood, and this will be still further increased by the frequent occurrence of double tumours. In the latter case the names to be assigned to the different forms will vary with the views of the individual making the classification. For instance, should a double intussusception that starts as a prolapse of the small gut through the ileocæcal valve be termed ileocolic-colic or ileocolic-ileocæcal? or, suppose an enteric intussusception becomes impacted in the valve, and then both valve and enteric intussusception become invaginated into the colon, is the resulting tumour to be called enteric-ileocæcal, or enteric-ileocolic-ileocæcal, or enteric-ileocolic-ileocæcal-colic, or enteric-ileocæcal colic? Such compound names are made up on the assumption that the last intussusception to be reduced is the first to be formed, but I do not know if there is any proof that this is the case.

Even in the case of single tumours there are difficulties. Thus: What is the proper designation of an intussusception that starts in the caput coli and eventually involves the small

* *Annals of Surgery*, May, 1903.

gut? Is it cæco-colic, or cæco-ileocæcal, or colic-ileocæcal? Again, suppose the appendix is found to be invaginated after the rest of the intussusception has been reduced, how is such a tumour to be named? In fact, any attempt to name all possible forms of intussusception can only lead to confusion.

I would suggest that instead of complicating the nomenclature it would be better to simplify it, and call all intussusceptions, involving large and small gut, entero-colic, and indicate whether they are double and where they started. Although the ileo-cæcal variety is rarer than the existing tables make out, my own series seems to show that it is still frequently to be met with as a single intussusception.

The observations on which this paper is founded were made with the suspicion that compound tumours were fairly common, and I was careful to observe the state of the bowel at the end of the reduction. In this series there occurred no example of an enteric or simple ileocolic intussusception. The latter, when it was met with, was only found after the main intussusception had been reduced. An ileocolic intussusception must always be of small extent, since the amount of small gut prolapsed through the ileo-cæcal valve must be limited by the length of the mesentery of the small gut, and it therefore stands to reason that the length of small gut involved cannot be more than double the length of the mesentery; as a matter of fact, it seems to very rarely reach even this extent. In this variety the increase in length is made at the expense of the entering tube; in this particular, and in that the most advanced portion is not constant, it differs from other intussusceptions, and is really more of the nature of a prolapse than a genuine intussusception.

Diagnosis.—This as a rule is sufficiently easy, since the occurrence of the usual history and symptoms with the presence of a tumour in the abdomen or rectum renders the diagnosis practically certain. It is indeed on the presence of the tumour that we must rely to differentiate this disease from some forms of enteritis. My own experience has led me to the belief that a tumour can in by far the greater number of cases be felt without difficulty, and should there be any doubt an anæsthetic will at once settle the question. The importance of giving an anæsthetic when in doubt, instead of waiting, has been insisted upon by Mr. F. C. Wallis,* and I should like to add my testimony to his. On the other hand,

* *Lancet*, June 11, 1904.

I have known a tumour confuse the diagnosis in two cases under my care. In one the tumour turned out to be a mass of glands near the cæcum, and in the other the tumour was a long dependent lobe of the liver.

Causation.—Of the sixteen cases in which a definite statement is made, there was only a history of improper feeding in three instances. In every instance the patients were well-nourished healthy children; they were not rachitic, nor did their appearance suggest improper feeding. Indeed, it seems probable that the success of the operative treatment may largely depend on the fact that the general good health of the patients enables them to combat successfully the shock of abdominal section. This series therefore throws no light, except of a negative character, on the causation of intussusception.

Duration before Operation.—The extremes were seven hours and four days. In no instance, with the exception of two cases in which resection was performed, and in which there was a possibility respectively of a fourteen and twenty-one days' history, was there any relation between the duration of the symptoms and the difficulty of reduction, neither could I trace any relation between the nature of the intussusception and the facility or otherwise of the reduction.

Treatment. — This may now be stated to be primary abdominal section. Inflation, except as an auxiliary measure to reduce the tumour out of the pelvis, as advised by Mr. Kellock, is now abandoned. If further proof of the inefficiency of inflation were needed, case 6 would furnish it. In this case inflation was tried three times, and on each occasion the tumour reappeared.

The site of the incision is open to discussion.

If the intussusception has reached the pelvis, no one incision will be the most convenient at all stages of its reduction. The site of the abdominal opening must therefore be largely determined by the fact that it is necessary to perform the last stages of reduction with the tumour in sight. The incision adopted in this series was one through the right rectus muscle; its length was about 3 in. and its centre lay $\frac{3}{4}$ in. to the right and below the umbilicus. This situation lies nearly at the centre of the circle traced by an intussusception, but is sufficiently near the normal site of the cæcum to permit the tumour to be easily brought out at the end of reduction, if not during the whole course. I have found that it is often extremely difficult to keep the tumour in sight while the re-

duction is being effected at the splenic and hepatic flexion of the colon.

The following was the technique adopted in this series: Directly the peritoneum was opened the end of the tumour was brought out of the wound and the reduction accomplished under the eye. If the tumour lay in the pelvis, it was necessary, occasionally, to reduce a few inches within the abdomen until the lower end came easily under the wound. No attempt was made to retain the intestines in the abdomen. If they escaped—and they did so in the majority of cases—they were simply covered up with a warm sponge. In fact, the intestines were not the object of any special solicitude. The point aimed at was rapidity of operation. The average time taken in eleven cases in which a note was made was 14 minutes; in this list are included a resection of gut and the excision of a gangrenous appendix.

Suture.—The incision through the rectus muscle facilitates rapid suture since the layers are quite distinct in this situation. The following were the methods adopted:

Suture in one layer	4
„ in three layers	9
„ through all layers and separate suture of anterior rectus sheath . . .	6
Not stated	1
	20

The material used was, as a rule, fine silkworm gut and, in a few cases, fine silk.

Of the nine cases sutured in three layers, two burst open and required re-suture; one of these ended fatally. The case that recovered was re-sutured by Mr. Llewellyn at the East London Hospital for Children.

This want of union in young children is a factor that must be reckoned with, and is best avoided, I think, by using deep sutures through all the coats and separate suture of the anterior rectus sheaths. The cause of the reopening of the wound is the failure of reaction and the sloughing of the delicate tissues in the embrace of the sutures, when only thin layers are included.

I have within the last six months seen the scar in all the successful cases, and in every instance they were perfectly sound. This fact tends to show that as regards ultimate results the method of suture is immaterial.

Results of the operations.—The time that elapsed between the

operation and the advent of death showed that the fatal result was not directly due to the effects of the operation. The intervals were: (1) Six days; (2) six days; (3) ten days; (4) few hours.

The causes of death were: (1) Bursting open of the wound; (2) thrombosis of cerebral veins (resection case); (3) gangrene of cæcum and inanition; (4) shock; extensive resection of nearly the whole of the large gut.

Mr. Barker, who has had great experience of these cases, lays special stress on reducing the intussusception within the abdomen in order to reduce shock. This series seems to show that after all too much stress can be laid on this point, as no attempt was made to keep the intestines in if they insisted in coming out. It seems to me that much valuable time may thus be lost. The two methods at any rate show equally good results.

The Appendix.—One interesting point in the series was the difference in the appearance of the appendix. In five instances this organ was normal in colour, though the intestine in its immediate neighbourhood was black from congestion. It did not appear that the variety of intussusception had any relation to the state of this organ.

After history.—With three exceptions I have traced all the successful cases.

In all the children are healthy and well, except in the following case, where attacks of vomiting occurred at times. This child succumbed to acute strangulation by a band in June, 1904.

A. W. B. (Case 1) suffered from attacks of vomiting, for which he was readmitted to hospital. With simple treatment the attacks subsided and as nothing abnormal was noted in the abdomen no operative measures were proposed. In June, 1904, the child (then æt. 4) was admitted for acute intestinal obstruction. The obstruction, due to a band passing from the mesentery to the bowel wall, was relieved, but death ensued in forty-eight hours.

It is remarkable that a child æt. only 16 weeks and extremely ill should have survived a cœliotomy for the reduction of an intussusception, while at the age of 4 it should have succumed to a like operation for the relief of acute obstruction by a band. It certainly serves to show how very grave a disease is acute obstruction by a band. This fact has been emphasised by Mr. Makins, who believes that but little im-

provement in the mortality of this disease has taken place of late years.

The fall in the mortality.—In my series there were twenty cases and four deaths, which gives a mortality of 20 per cent. If the cases in which a resection was performed are excluded, the mortality falls to 11·11 per cent. Taking the cases under 1 year of age, there were sixteen, with three deaths —mortality 18·75 per cent., or, excluding the case of resection, 13·33 per cent.

From Sargent's tables (*St. Thomas's Hospital Reports*, 1900), I find that from 1892 to 1901 there were sixty-two cases of intussusception in children under one year, which were treated by cœliotomy (inflation had been tried in some). There were 38 deaths, or a mortality of 61·29 per cent. (If all the cases under 1 year are taken into account the numbers are seventy-two cases, and the mortality 61·11 per cent.)

Barker* gives the following figures from cases that occurred in University College Hospital from 1892 to 1896 (both years included): Under 1 year, fifteen cases—mortality 40 per cent. Under 1 year and treated by cœliotomy (either primary or after inflation) fourteen cases—mortality 42·86 per cent.

D'Arcy Power† in 1898 stated that the mortality of cases (not necessarily under 1 year) operated on within forty-eight hours of the onset was 22 per cent., and expressed the belief that this would be lower in the near future.

The improvement that has taken place is well shown in the following table of cases treated at St. Thomas's Hospital (Sargent). All cases under 1 year (no matter how treated).

	Died.	Cured.		Died.	Cured.
1892	3	1	1898	4	2‡
1893	2	0	1899	6	8
1894	1	1	1900	6	6
1895	4	1	1901	7	7
1896	5	1		—	—
1897	6	1		44	28

Total, 72. Mortality, 61·11.

In the last three of the above years there were forty cases, with 19 deaths, or a mortality of 47·5 per cent.

* *Clin. Soc. Trans.*, vol. xxxi.

† "Some Points in the Anatomy, Pathology, and Surgery of Intussusception."

‡ Cœliotomy (primary) as a routine measure.

Intussusception, like appendicitis, seems to be on the increase, at any rate as regards those admitted to hospital.

The following figures are from St. Thomas's Hospital:

Year (periods of 5 years).	Number of cases of intussusception	Total number of admissions to St. Thomas's Hospital.
1879–1883	10	20,016
1884–1888	10	23,103
1889–1893	12	25,338
1894–1898	40	29,493
1899–1903	90	31,547

From this it will be seen that the number of intussusceptions has increased ninefold, while the total admissions, all departments included, has only increased by one half. The increase is therefore actual and relative.

In conclusion, and as showing well the strides that have been made in treatment, I cannot do better than quote one or two passages from a book written by a great authority on this subject in 1884: "The administration of opium is absolutely essential in these cases; . . . there are substantial reasons for believing that under the influence of opium, administered early, an invagination may undergo cure by spontaneous reduction. . . . Perceiving, as is probable, that no marked improvement follows, . . . the next measure is an attempt to reduce the invagination by means of enemata or insufflation. These modes of treatment have met with very encouraging success, and are worthy of a patient trial. . . . Should these measures fail, there are substantial reasons for recommending an immediate laparotomy, especially in the young."

In twenty years, then, cœliotomy has risen from a last resource to a primary measure.

Tables.

Mr. Cuthbert Wallace's Paper on A Series of Intussusceptions in Children.

No.	Name.	Sex.	Age.	Duration to operation.	Situation of tumour.	Method of feeding.	Nature of intussusception.	Method of suture.
1	A. W. B.	M.	16 weeks	7 hours	Left iliac fossa	Not stated	Ileo-cæcal	1 layer
2	A. S.	M.	5 months	10 hours	Umbilical	"No improper food"	Enteric—ileo-cæcal	Not stated
3	F. W.	M.	20 weeks	2 days	Present; situation not stated	Not stated	Ileo-cæcal	3 layers
4	F. C.	F.	33 months	6 days ? 3 weeks	Left iliac fossa, also protruded from anus	Nothing wrong noted	Ileo-cæcal	1 layer
5	J. B.	M.	17 months	3 days	Left loin, also felt in rectum	Breast, also bread and butter	Ileo-cæcal	Deep and rectus sheath sutures
6	L. L.	F.	7 months	30 hours	Left iliac fossa	Breast, occasional bread and butter	Enteric—ileo-cæcal	Deep and ant. rectus sheath sutures
7	H. R.	M.	8 months	4 days	Right hypochondrium	Breast	Ileo-cæcal	3 layers with 1 deep suture
8	A. H.	F.	3 months	36 hours	Left lumbar, felt in rectum	Breast	Ileo-cæcal	3 layers with 2 deep sutures
9	C. A.	F.	6 months	2 days	Left loin, felt in rectum	Breast and bottle	Ileo-cæcal	Deep and ant. rectus sheath sutures
10	J. R.	M.	5 months	26 hours	Splenic region	Breast	Enteric—ileo-cæcal	3 layers and 1 deep suture
11	S. B.	M.	5 months	18 hours	Umbilical region	Breast. Had a piece of mackerel	Ileo-colic—ileo-cæcal	3 layers

Duration of operation.	Reduction.	Result.	Remarks.	Year.	Ultimate history.
Under 25 minutes	Easy	C.	Child apparently moribund before first operation	1898, May	Jan., 1904.—Scar sound. Had attacks of vomiting, cause not to be made out. Died of acute strangulation by a band, June, 1904.
10 minutes	Final part of ileo-cæcal difficult	C.	Reduction by inflation had failed. Small enteric tumour remained after main reduction	1899, May	Well at end of six months. Jan., 1904.—Not traced.
7 minutes	Difficult at cæcum	D.	Wound found burst open on 4th day. Re-sutured	1899, Jan.	*P.M.*—Cæcum still infiltrated with blood. Died on 6th day.
10 minutes	Impossible	D.	Reduced to hepatic flexure, when outer coat was found to have sloughed in a linear manner for the extent of three quarters of circumference of gut. Excision. Artificial anus. Food taken well. Bowles acted	1899, April	Died on 10th day. *P.M.*—Thrombosis of cerebral veins. No peritonitis.
10 minutes	Easy	C.	Pulse 160 on admission, abdominal facies. Tumour palpable only under anæsthetic	1901, July	Well in October, 1903.
10 minutes	Difficulty at cæcum	C.	Inflated three times but recurred. Enteric tumour measured 2½ inches. Appendix normal in colour, though cæcum was black	1901, July	Not traced.
13 minutes	Difficult near cæcum	C.	Appendix quite normal in colour. Inflation had failed	1901, Nov.	Well in October, 1903.
13 minutes	Difficult	C.	Tumour only felt under anæsthetic. Appendix normal in colour though cæcum was black	1901, Dec.	Quite well. Scar sound. Feb., 1904.
Not stated	Easy	C.	Tumour could be felt in rectum	1901, April	Jan., 1904.—Quite well. Scar sound.
9 minutes	Difficult at cæcum	C.	Appendix only slightly congested	1902, Nov.	Jan., 1904.—Quite well. Scar sound.
27 minutes	Very difficult indeed	D.	Appendix normal at base, but sloughing at apex, excised. 4 inches of ileum through valve. Last 2 inches of ileum had a thick patch of infiltration on wall opposite mesentery, suggestive of a tumour, incision showed only blood in tissues	1903, April	Child died of marasmus. *P.M.* — Cæcum and several inches of colon black and almost gangrenous. Death on 6th day.

No.	Name.	Sex.	Age.	Duration to operation.	Situation of tumour.	Method of feeding.	Nature of intussusception.	Method of suture.
12	E. A.	F.	5 months	18 hours	Umbilical region	Breast	Ileo-colic—ileo-cæcal	3 layers
13	P. S.	F.	21 months	2 days	Splenic region	Not stated	Colic	3 layers
14	W. F.	M.	4 months	12 hours	Splenic region	Breast	Ileo-colic—ileo-cæcal	3 layers
15	S. P.	F.	3 months	28 hours	Splenic region	Breast	Enteric—ileo-cæcal	Deep and ant. rectus sheath sutures
16	R. W.	M.	8 months	8 hours	Left loin	Breast	Ileo-cæcal	Deep and ant. rectus sheath sutures
17	M. R. F.	F.	2 years	2 days	Left loin	Nothing abnormal	Ileo-cæcal	3 layers
18	A. W. P.	M.	5 months	14 days or 30 hours	Left loin	Breast, some bread	Ileo-colic—ileo-cæcal	Deep sutures
19	R. S.	M.	6 months	25 hours	Umbilical region	Not stated	Ileo-colic—ileo-cæcal	Ant. sheath and deep sutures
20	L. C. W.	M.	9 months	1 week	Left loin	Breast till 8 months, then little bread and butter	Ileo-cæcal	Deep

Cases 20. Males 12. Females 8.

Ileo-cæcal . . .	10 or 50	per cent.
Enteric-ileocæcal .	4 or 20	„
Ileocolic-ileocæcal .	5 or 25	„
Colic	1 or 5	„
	20 or 100	„

Duration of operation.	Reduction.	Result.	Remarks.	Year.	Ultimate history.
20 minutes	Ileo-colic difficult	C.	Ileum through valve for space of 3 inches	1903, May	Jan., 1904.—Quite well. Scar sound.
Not stated	Easy	C.	Dimple in the transverse colon at starting-point of tumour	1903, Aug.	Jan., 1904.—Quite well. Scar sound.
Not stated	Easy	C.	6 to 8 inches of small gut through the valve, last 2 inches deeply congested. Wound found burst open, re-sutured	1903, Sept.	Well some months after operation, 1904.
Not stated	Easy	C.	Small enteric separated by a distinct interval from the valve	1903, May	Well some months after operation, 1904.
Not stated	Difficult at cæcum	C.	Cæcum dimpled; apparently this was the starting-point	1903, Nov.	Jan., 1904.—Quite well. Scar sound.
Not stated	Difficult	C.	Large dimple on cæcum that would not be everted. Appendix normal in colour	1903, Feb.	Not traced.
20 minutes	Extremely difficult	C.	Cæcum burst open in attempt to reduce ileocolic portion. Inner middle layers extruded and reduced like a paraphimosis. Cæcum sutured.	1904, Feb.	
15 minutes	Easy	C.	Appendix normal in colour	1904, March	
20 minutes	Impossible	D.	Gut ruptured just below splenic flexure. Not gangrenous but extremely friable. Resection. Artificial anus	1904, April	Death in a few hours.

Died 4. Mortality 20 per cent.; or excluding two cases of resection 11·11 per cent.

Under one year of age:
Mortality 18·75 per cent., or excluding the case of resection 13·33 per cent.

XI.—*Cases of Intussusception in Children treated by Laparotomy.* By C. H. Fagge. *Read December* 9, 1904.

The eighteen cases of intussusception in children on which this paper is founded are all which have come under my care at either the Evelina Hospital for Sick Children (thirteen), or at Guy's Hospital (five.)

I have not operated on any case as one of intussusception which has not proved to be intussusception, nor have I met with a case in which an intussusception has been overlooked.

So many writers of repute have, for the past seven years, urged the advantage of laparotomy over other methods of reduction, that its advisability must now be agreed upon; but well-recognized lines of treatment, especially if simple and non-operative, die hard, and so inflation will only be finally given up after many more successful laparotomies have been recorded.

Ætiology.—Of the cases under 1 year, fourteen in all, ten were stated to have been entirely breast-fed, and all the babies were decidedly above the average of hospital patients in size and degree of nourishment.

Presence of a tumour.—In sixteen of the eighteen cases a tumour was felt either through the abdominal wall or *per rectum*, and several times the value of bimanual examination, as suggested by Mr. Eve (*Clinical Journal*, 1899), was demonstrated; in fifteen of these sixteen cases diagnosis was easy, and treatment was prompt. In one case (No. 16), on the history of some weeks' abdominal pain, with occasional attacks of vomiting, a diagnosis of tuberculous peritonitis was made, the tumour being regarded as matted omentum and intestines, and in consequence of this, operative interference was undertaken too late; this was probably a chronic intussusception, in which the acute exacerbations were caused by an increase of the invagination.

Both the cases in which no tumour was found ended fatally; in one (No. 4) the symptoms were not typical, and operation was delayed for twenty-four hours after her admission. Her symptoms before death were most peculiar, and were suggestive of some cerebral lesion rather than of any result of

the intussusception; unfortunately, no *post mortem* could be obtained. In the other case (No. 11) the abdominal distension, and the position of the intussusception, which was enteric, no doubt were responsible for the failure to detect a tumour.

Yet, while a tumour was so frequently present, my cases well emphasize the variability of its position and the value of an examination under an anæsthetic in locating it. In fact, the observance of routine examination under an anæsthetic has several times led to the detection of a tumour in a case which might otherwise have been regarded as one of enteritis; unfortunately, in Case No. 4 this precaution was neglected.

Duration before operation.—All writers agree that there is no exact relation between duration and ease of reducibility; but of those in which the time is given, all of less than forty-eight hours' duration were reducible, though with varying ease, which strikingly bears out Gibson's (*Annals of Surgery*, 1900) contention that after the second day the ease of reducibility markedly diminishes; for he reckons that on the first day 94 per cent., on the second day 83 per cent., and on the third day 61 per cent. are reducible.

Variety of intussusception.—After Mr. Corner's exhaustive paper (*Annals of Surgery*, 1903), and as Mr. Wallace has fully dealt with this part of the subject in his paper, I do not propose to say much about the varieties of intussusceptions met with. It seems now probable that single ileocæcal intussusceptions are not as common as earlier writers—Sargent (*St. Thomas's Hospital Reports*, 1900) and Eccles (*St. Bartholomew's Hospital Reports*) considered; yet, though I have since my first operation carefully looked for double intussusceptions, ten, at least, of my eighteen cases were of the single ileo-cæcal type. The eleventh single intussusception was enteric (No. 11), and was of interest in that the ileum at the apex of the intussusception was so much narrowed as to suggest the possibility of a congenital stricture at that spot having given rise to the intussusception. A second point worth mention in this case was a persistent Meckel's diverticulum; this was some distance above the intussusception and clearly had never been involved.

I can only record seven double intussusceptions: Colic-ileocæcal, 3; ileo-colic-colic, 2; enteric-ileocæcal, 2.

My early acquaintance in April, 1900 (Case No. 2), with the cæcal variety of Mr. Eve (*Clinical Journal*, 1899), described by Mr. Corner as colic-ileocæcal, led me to look for

this form again, but only two other examples (Nos. 14 and 17) were met with, making 19 per cent. of the sixteen cases.

In Case No. 7 there was possibly a triple intussusception, for the ileum was prolapsed into or through the ileo-cæcal valve, and there was a well-marked and apparently independent invagination of the caput coli some distance to the right of the valve, so that this may be colic ileocolic-colic, and not ileocolic-colic, as I have regarded it in my list.

I do not attach great value to a minute division of intussusceptions into many varieties, and at an operation completed as rapidly as possible it is obviously most difficult to distinguish between forms so nearly related as ileocolic-colic and enteric-ileocæcal (Corner.)

Treatment and technique.—In seventeen of my cases primary laparotomy was undertaken at the earliest possible moment; in the eighteenth (No. 9) inflation had been twice tried and had failed before I saw the case. Except in Cases 11 and 16, my incision was always made on the right side of the middle line, with the centre at or below the level of the umbilicus; in the majority it was through the right semilunar line; but recently I have cut vertically over the right rectus, separating its fibres, thinking that by this means a firmer scar might be obtained.

I have not made use of the combined method of inflation with laparotomy (Kellock), which, in my opinion, besides the time necessary for its performance, has the great disadvantage that it increases the intra-abdominal contents, and therefore will militate against the replacement of the already distended intestines within the abdomen. I have found that a second assistant's finger, introduced *per rectum* promptly and gently, reduces the intussusception usually as high as the iliac colon, where it can be easily dealt with by the operator from the abdominal cavity.

Until the reduction has reached the ascending colon it is carried out partly out of sight; then the intussusception is delivered from the abdomen, and the remainder of the procedure carefully watched.

Escape of the intestines during the manipulation of the intussusception is, I feel sure, to be avoided whenever possible, and, in fact, often the chief difficulty is experienced and much loss of time is occasioned in returning the escaped bowel within the peritoneal cavity; further, I feel sure that the dragging of the dependent intestines upon the mesenteric plexuses increases shock. When this does happen and the

bowel is much distended, I have found it most useful for the assistant to strongly lift up and separate the two edges of the wound with Spencer Wells' forceps, when the bowel can be paid back coil by coil into the abdomen.

Unless the condition of the patient was extreme, the abdominal wall was always united by three layers of sutures, silk in the early cases and catgut recently being used for the buried sutures.

Results.—Six cases (Nos. 3, 10, 11, 15, 16, and 18) died between four and twenty hours after the operation from shock and toxæmia; of these, in Nos. 11, 15, 16, and 18, this was clearly due to the resection of the bowel. One (No. 10) was so ill that nothing more than the formation of an artificial anus could be attempted.

As there was no *post mortem* in Case 4, I cannot suggest the cause of death on the fourth day; when the abdomen was opened a second time, it was thought that the intestines might not have recovered from the strangulation, but everything was found to be perfectly healthy, and there was no obvious cause for her condition.

I do not propose with my small number of cases to dwell on statistics; my immediate results show a mortality of 39 per cent. (seven deaths in eighteen cases); if we include two other babies (Cases 5 and 6) who were readmitted within a few days of their discharge, and whose death was indirectly a result of the intussusception, the mortality increases to 50 per cent.; both these died twenty-three days after the laparotomy, which could have had no relation to the ultimate result.

I have perhaps been unfortunate in the large proportion of irreducible and gangrenous intussusceptions (five cases) that have come under my notice; for, excluding these, the mortality of two out of thirteen laparotomies works out at 15·4 per cent.

I am sure everybody will agree that no mechanical means alone could reduce an intussusception found to be irreducible or gangrenous at a laparotomy; and yet I know of no statistics embracing a large number of cases treated alone by inflation which give a mortality even as low as 50 per cent.

In the two cases (Nos. 5 and 6) who were re-admitted for diarrhœa, it is probable that the intestines, though easily reduced, did not return to their normal condition. In support of this idea, the notes of the *post mortem* on No. 5 are most interesting, for here the reduction was peculiarly easy and the intestines at the time of operation apparently only slightly

strangulated. It is therefore probable that the same result would have followed reduction by mechanical means, which suggests that intussusception may be followed by remote as well as immediate risks, and that the recovery of the child from his acute illness may be more apparent than real. It would be interesting to know whether the ultimate results in a large number of cases treated by different methods confirms this suspicion of vulnerability in children who have recently suffered from intussusception.

May I venture to suggest for discussion the treatment of irreducible intussusception.

Here I think we must separate simple irreducible cases from those which, besides being irreducible, are also gangrenous. Thinking over my five irreducible cases (Nos. 10, 11, 15, 16, and 18), I am in agreement with Gibson (*Annals of Surgery*, 1900) in decidedly preferring resection in gangrenous cases to the formation of an artificial anus, for, as Gibson points out, the latter does not remove the infective intestine.

In non-septic irreducible cases either the formation of an artificial anus or a longitudinal division of the ensheathing and returning layers may be undertaken, and I intend in my next case to adopt the latter course, following it, if necessary, by a modified resection.

In the four cases with gangrenous bowel resection was carried out, in three (Nos. 11, 15, and 16) by enterectomy between Lane's clamps and end-to-end union by simple suture in two layers, the inner passing through all the coats and the outer through the peritoneal and muscular coats only. In the eighteenth case the apex of the intussusception was exposed by a longitudinal incision through the ensheathing layer and cut off, the cut ends of the intestine being united by Maunsell's method.

Gibson (*loc. cit.*) quotes sixty-one cases of irreducible or gangrenous intussusceptions, of which forty-nine died, and the fact that there was only one resection for a gangrenous intussusception among the twelve recoveries is a strong argument for inquiry as to the best line of treatment in these cases.

Tables.

Mr. C. H. Fagge's Cases of Intussusception in Children treated by Laparotomy.

No.	Name.	Sex.	Age.	Date.	Feeding.	Symptoms.	Position of tumour.	Time of operation aft. onset.
1	L. C.	F.	3½ months	April, 1900	Breast	Sudden onset, crying out and vomiting. Blood passed 10 hours after onset. Tumour felt in abdomen	Right hypochondrium	21 hours
2	C. W.	F.	3½ months	April, 1900	Breast	Sudden onset, screaming, refused food, sick. Blood 6 hours after onset. Abdomen rigid and distended. Nil on R.E., but tumour felt bimanually under anæsthetic	Right inguinal region, just above pelvic brim	45 hours
3	A. E.	M.	5 months	Dec., 1900	Breast	Sudden onset, screaming. Blood passed 5 hours after onset, vomiting. Tumour felt, and on R.E. apex of intussusception 1¼ in. from anus	Left iliac region	33¼ hours
4	L. R.	F.	9 years	March, 1901	—	Vomiting, pain under right rectus; bowels open, no blood. Sent in diagnosed as intussusception. Nothing found even under anæsthetic, very sick	No tumour	3 days
5	E. N.	M.	3 months	July, 1901	Breast	Sudden onset, crying, and soon passed blood and mucus, vomited. Tumour felt. R.E. nil	Right lumbar region	15 hours
6	M. G.	F.	6 months	Sept., 1901	Breast	Sudden onset, vomiting, crying, passed blood and mucus. Tumour felt. R.E. revealed nil, but blood and mucus was on finger; nothing on bimanual examination	Right lumbar and hypochondriac regions	29 hours
7		M.	8 months	Jan., 1902	Breast	Strained and passed normal stool, when "prolapse" came down, which doctor reduced, after which blood and mucus were passed, and "prolapse" came down again	Projecting from anus	30 hours

Nature.	Suture.	Duration of operation.	Result.	Remarks.	Ultimate result.
Ileo-cæcal	2 layers	40 mins.	Discharged	Reduction difficult. Peritoneum torn in several places and sutured with silk. Child was very ill, with vomiting for 30 hours after operation	September, 1901.—I opened a post-pharyngeal abscess. December, 1904.—Quite well, scar firm
Colic-ileo-cæcal	1 layer	Not stated	Discharged	Reduction very easy. Great difficulty in returning intestines which had escaped	—
Ileo-cæcal	1 layer	35 mins.	Died	Reduction extremely difficult. Peritoneum and muscular coat much torn; sutured. Intestines returned with difficulty. Death 20 hours after operation	No *p. m.*
Ileo-cæcal	3 layers	Not stated	Died	Child was not admitted until 2 days after onset; and when seen next day I diagnosed "obstruction." Intussusception was found with difficulty; easily reduced. Much improved after operation, but on 4th day became comatose and moaned. I again opened abdomen and found nothing	No *p. m.*
Ileo-cæcal	3 layers	About 20 mins.	Discharged	Reduction easy. Child did well for 8 days, when diarrhœa began: this improved, and he was discharged to be fed by mother, but he was brought back next day very ill and died 24 days after operation	*P. m.* — No peritonitis; cæcum, ascending colon and end of ileum greyish and very friable and much collapsed. Catarrhal enteritis
Enteric-ileo-cæcal	3 layers	18 mins.	Discharged apparently well	Was not admitted until 24 hours after onset. Reduction easy. Last 2 ins. of ileum reduced last	Re-admitted next day with diarrhœa and vomiting, and died 11 days later. No *p. m.*
? Ileo-colic-colic. ? Colic-ileo-colic colic	3 layers	22 mins.	Discharged	Reduction very easy after all the part in rectum had been reduced by a finger within rectum	December, 1904.—Quite well. Scar firm.

No.	Name.	Sex.	Age.	Date.	Feeding.	Symptcms.	Position of tumour.	Time of operation aft. onset.
8	W. M.	M.	6 months	June, 1902	Not stated	Blood passed, vomiting, and tumour felt	Not stated	—
9	J. D.	M.	9 months	Aug., 1902	Not stated	Sudden onset, vomiting, blood passed, tumour felt	Umbilical region	9 hours
10	A. G.	M.	3 months	Aug., 1902	Breast	Sudden collapse, vomiting, blood and slime passed, tumour felt. On R. E. no tumour felt, but blood on finger	Left side, mainly in left iliac region	53 hours
11	R. B.	M.	15 years	Sept., 1902	—	Pain in abdomen, vomiting. B.o. only once since onset (5 days), and then blood was passed. Some distension	No tumour	5 days
12	A. D.	M.	11 months	Dec., 1902	Breast and other food	Sudden onset of vomiting, tumour easily felt. R. E. disclosed apex 1½ in. from anus	Left side, from left hypochondrium to left iliac fossa	9½ hours
13	C. R.	F.	5 months	Feb. 1904	Breast	Sudden onset, crying, straining, persistent vomiting, passage of blood and slime. No tumour felt until anæsthetic given, when a transverse tumour was found	Umbilical region	32½ hours
14	E. C. M.	M.	4½ months	March, 1904	Not stated	Crying, passed mucus and blood, tumour felt under anæsthetic	Not stated	5½ hours
15	B. M.	M.	3 years	April, 1904	—	—	—	—
16	E. B.	F.	9½ years	April, 1904	—	Has been ill for six weeks with abdominal pains and vomiting, was treated at another hospital, and admitted to Evelina three weeks before operation. Mass felt around umbilicus. There was only occasional vomiting until three days before operation, after which it was frequent. Tumour unaltered. Abdominal pain almost constant. Bowels irregular. No blood passed	Umbilical	? 3 weeks, ? 3 days

Nature	Suture.	Duration of operation.	Result.	Remarks.	Ultimate result.
Ileo-cæcal	—	—	Discharged	Report missing. Notes from observers	Re-admitted in Sept., 1902, and died of broncho-pneumonia, cæcal region normal.
Ileo-cæcal	3 layers	15 mins.	Discharged	Two attempts at inflation with milk failed to cause disappearance of tumour. Reduction easy	—
Unreduced, at *p. m.* found to be ileo-cæcal	Not sewn up	Under 20 mins.	Died	Intussusception could not be reduced, so a Paul's tube was tied into ileum and the intussusception left outside	Died 5 hours later. At the *p. m.* Dr. French could not reduce the intussusception.
Enteric	3 layers	45 mins.	Died	Peritonitis. Resection of 18 ins. of small intestine, which was gangrenous and perforated, end-to-end anastomosis with simple suture. Meckel's diverticulum present; not involved	Death 4 hours after operation. Resection 6 ins. from ileo-cæcal valve
Ileo-cæcal	3 layers	35 mins.	Discharged	Reduction difficult and prolonged	December, 1904.—Is delicate.
Colic-ileo-cæcal	3 layers	17 mins.	Discharged	Reduction easy. Well-marked cæcal dimple	December, 1904.—Seems well, but still passes blood. Scar firm.
Ileo-cæcal	3 layers	Under 30 mins.	Discharged	Reduction easy; suture of abdominal wall took a long time because of tendency of bowels to prolapse	—
Enteric-ileo-cæcal	3 layers	About 45 mins.	Died	Irreducible. Enteric intussusception resected and gut united by end to end anastomosis with simple suture	Died 6 hours later. *P. m.*, early peritonitis
? Ileo-colic-colic. ? Ileo-cæcal	3 layers	Under 60 mins.	Died	The case was regarded as tuberculous peritonitis; when I saw her just before operating, she had pulse 160, abdominal facies, was very restless, constantly sick, abdomen extremely tender and rigid: indicating "obstruction." The intussusception extended into descending colon, and the part in the ascending colon could not be reduced, so it was resected and end-to-end anastomosis done	Died 5½ hours after operation. *P. m.*, no peritonitis.

No.	Name.	Sex.	Age.	Date.	Feeding.	Symptoms.	Position of tumour.	Time of operation aft. onset.
17	C. E. U.	M.	11 months	June, 1904	Breast	Sudden vomiting, much crying with pain. Tumour felt. On R. E. nothing found	Right hypochondrium and epigastrium	8½ hours
18	E. S.	M.	8 months	Dec., 1904	Breast	Passage of blood. Severe abdominal pain; no vomiting. Treated 3 weeks for dysentery	Not felt in abdomen, easily felt *per rectum*	3 weeks

Cases 18. Males 12. Females 6.

Ileo-cæcal	10
Colic-ileocæcal	3
Enteric-ileocæcal	2
Ileocolic-colic	? 2
Enteric	1

Nature.	Suture.	Duration of operation.	Result.	Remarks.	Ultimate result.
Colic-ileo-cæcal	3 layers	20 mins.	Discharged	Reduction easy. Rapid recovery, but on 13th day wound burst and small intestine and omentum prolapsed. Under anæsthetic gut returned and wound re-sutured in one layer	Was re-admitted with whooping-cough, of which he died, September, 1904.
Ileo-cæcal	3 layers	45 mins.	Died	Abdomen very much distended and contained turbid fluid; last 3 ins. could not be reduced and ensheathing layer torn, so this was opened and tumour exposed and removed. The gut was joined by Maunsell's end-to-end anastomosis	He died 5 hours later. *P. m.*, early peritonitis.

Died, 7.

Mortality . . . . . 39 per cent.
Or excluding irreducible cases . . 15·4 „
Under 1 year of age: Mortality . . 21·4 „
Excluding irreducible cases: Mortality . 8·3

XII.—*Acute Ascending Paralyses in Cases of Chronic Cystitis.* By T. J. Walker, M.D. *Read January 13, 1905.*

In the comparatively limited field offered in my private practice, general and consulting, I have met with three cases of long-standing chronic cystitis, which terminated fatally by acute ascending paralysis of a most malignant type.

Although urinary paraplegia has been generally recognised by the profession for seventy years, since 1833, when Stanley directed attention to the malady in a communication to the Medico-Chirurgical Society, I can find no allusion by authors dealing either with urinary diseases or with Landry's paralysis, to the occasional association of the latter ailment with chronic cystitis. Stanley gives a short note of the case of a man admitted with gonorrhœa and phymosis into St. Bartholomew's Hospital under Dr. Erle, who died suddenly three weeks after admission and twelve hours after paraplegia had set in; but Mr. Stanley expressly mentions that the paralysis was not higher than the waist, and as the man suddenly fell back in his bed and died, the case was certainly not identical with those which occurred in my practice.

Saundby gives the case of a man æt. 60, admitted into the General Hospital, Birmingham, who died four weeks after the onset of symptoms of a progressive paralysis; the disease here was possibly pathologically of the same nature, although not showing the same terrible rapidity in its course as did the cases of which I will now detail the salient points.

Case 1.—Thomas G., æt. 40, baker. When fifteen years old fell astride a rail and injured his perineum. Ten years later he came under my care for retention of urine and stricture of urethra, and was treated in the Peterborough Infirmary. The retention had come on gradually, the patient having gone about habitually with a distended bladder; he never completely lost the chronic cystitis, which did not inconvenience him greatly, and did not prevent him taking and working a baker's business. He seldom sought medical aid unless there was an acute exacerbation or some indication of the stricture requiring dilatation. In April, 1877, such an

exacerbation occurred, and Mr. G. had been under treatment by my partner (Dr. Kirkwood) and myself for nearly a month, when, on the morning of May 19, I was sent for and found that in the course of the previous night he had felt numbness in the feet and some loss of power in the legs. When I saw him there was paresis of the limbs up to the thighs; by the evening the paralysis had extended upwards to the arms and thorax, and the breathing was already embarrassed; the symptoms rapidly progressed, and death took place eighteen hours after their onset.

No *post-mortem* was made, but it seemed probable that some direct causal relation between the urinary disease and the rapidly fatal, almost fulminating, ascending paralysis must exist.

My notes of the case are very meagre, but to the best of my recollection the patient did not require the use of the catheter or bougies during this last illness.

Case 2.—W. R., æt. 62, farmer. Was first seen by me about the year 1877, when, in consultation with Dr. Elliott, of Oundle, I operated upon him for a stricture of the meatus of the urethra, for which he sought medical aid only, when the symptoms of distended bladder and cystitis, with painful micturition, made his life intolerable. A few years later, having come to live close to Peterborough, he came under my care, and I learned that he had never lost his cystitis.

On December 31, 1886, I was called to him on account of an acute exacerbation of his bladder trouble, accompanied with much malaise and some fever; the symptoms were not urgent, there was no difficulty in micturition, and no catheter was passed. I saw him again on January 2; he was somewhat better. He was not visited on January 3, but on the early morning of January 4 he experienced uncomfortable sensations and some muscular feebleness in the lower limbs, and at my visit, about mid-day, I found distinct indications of paraplegia; the sudden onset of the paralytic symptoms, with the history of long-standing cystitis, etc., and the feeble general condition reminded me at once of the case which I had seen ten years previously. I gave a prognosis which led to the patient promptly settling his affairs. On the next day the symptoms had made considerable progress, and early on the morning of January 6, forty-eight hours after the first indication of paralysis, the patient succumbed. A necropsy was not allowed.

Case 3.—This case was seen by me in consultation with the late Dr. Pilcher, of Boston, whose son, Dr. C. W. Pilcher, has furnished me with notes of the case.

W. H., æt. 67, a butcher, residing at Boston, had for many years suffered from chronic cystitis and for the three months preceding his death had been under Dr. Pilcher's care suffering from prostatic disease and cystitis requiring catheterisation; he had been confined to his bed for some time. On the morning of March 13, 1894, when he got out of bed, the patient appeared feeble and unsteady on his feet; he was catheterised as usual by Dr. Pilcher, and in the course of the day I was sent for. I saw the patient at about 8 p.m.; I noticed when I wished to feel his pulse that the movement of the hand and arm were impaired, and on further examining into the history recognised that I had another case of the same character as Cases 1 and 2. Already, only twelve hours after the onset of the symptoms indicating implication of the nerve centres, the paralysis had progressed upwards until the upper extremities were involved.

With my experience of the former cases I gave a prognosis of urgent danger, demanding the immediate settlement of the patient's affairs; this opinion was justified by Mr. H.'s death eighteen hours after the first onset of the paralytic symptoms. No necropsy was made.

Each of these three cases of progressive ascending paralysis is distinguished by the sudden invasion of the attack, by its terribly rapid progress, and by the fact that it occurred as the fatal termination of a chronic cystitis.

As to their pathology I am, unfortunately, able to give no certain information; they are recorded mainly for the benefit of those actively engaged in the prosecution of our art—those who, like myself, have little time or opportunity to advance our science.

I can recall only two other cases of this very acute type of ascending paralysis in which I have been consulted, so that my experience is that out of five cases of this malady three were associated with urinary disease; and yet by none of the authors whom I have been able to consult is this association referred to. Dr. Farquhar Buzzard, in the important paper on the pathology of Landry's paralysis, published in *Brain* last year, speaking of the associated morbid conditions met with sufficiently often to justify their mention, names only two—enlargement of the spleen and evidence of extensive pleurisy in the form of old and recent adhesions; but in the

light of my experience I cannot believe that the absence of recorded cases must be taken as a proof of the rarity of the association of Landry's paralysis with disease of the urinary organs: it is rather that, this association never having been recognised as causal in its nature, cases have not been published.

A very brief glance at the pathological history of the nervous affections known to be associated with disease of the bladder and at that of Landry's paralysis, will, I think, help to show the probability of the occurrence of cases such as those which I am reporting in which, as I believe, the one disease gave rise to the other. Although rarely alluded to by writers on diseases of the urinary organs, "urinary paraplegia" has, as I have already said, been recognised since Stanley, in 1833, directed attention to the malady, accounting for it in accordance with the pathological views of that date as a form of reflex paralysis; and even fifty-three years before this Troja (as quoted by Leyden) stated that violent inflammation of the kidney may produce an irritation of the nerves of that organ which may extend to the spinal cord, giving rise to paralysis and loss of sensation in the lower limbs. Gull, in a paper published in *Guy's Hospital Reports* for 1861, attacked the wavering faith in the nervous reflex view of the pathology of urinary paraplegia, contending that it was due to an inflammation of the cord which escaped detection in fatal cases owing to the imperfection of the examinations made at that time (1861). In this paper he enumerates under fifteen heads the characteristics of urinary paraplegia. The second, third, and fourth of these are: second, usually only lower limbs paralysed; third, no gradual extension of the paralysis upwards; fourth, usually paralysis incomplete. From which it is clear that every character of "the symptom complex" (Dr. Farquhar Buzzard), which we call Landry's paralysis, is wanting in "urinary paraplegia" as hitherto described. Leyden, in a clinical lecture on the subject of reflex paralysis, goes fully into the history and pathology of urinary paraplegia (German Clinical Lectures, New Sydenham Society), and he concludes that in a considerable number of cases an anatomical affection of the cord has been proved in the form of a myelitis which begins in a circumscribed lesion in the upper part of the lumbar enlargement; he assumes that the inflammation first passes to the nerves and induces in them a progressive neuritis which, as has been proved by experiment, may lead to myelitis; he concludes that this may, therefore, be looked upon as

the most natural and frequent mode of occurrence in paraplegia urinaria.

Mr. Hurry Fenwick, who gives a fuller consideration to the matter than any other of the writers on urinary diseases whom I have been able to consult, while he points out that it would be rash to throw over entirely the possibility of reflex paralytic symptoms in connection with urinary diseases, accepts the more recent view of the pathology of urinary paraplegia as expounded by Leyden and others. Beyond this stage the pathology of urinary paraplegia has not been advanced. I can find no record of bacteriological investigation in this malady.

Affections of the bladder associated with, but secondary to, disease of the nervous system have not, I think, any bearing on the question under consideration; therefore I now pass briefly to the history of Landry's paralysis, by which I mean acute ascending paralysis, such as that which proved fatal to my patients—a mortal malady which until now, as Farquhar Buzzard points out, is to us only a "symptom complex," not a disease.

Landry, in his original paper, published ten cases of ascending paralysis showing a pernicious and malignant character, differing from other cases of general progressive paralysis characteristic of well-known morbid conditions, in the essential point that there was in these cases an absence of every appreciable nervous lesion. This, which he regarded as the essential characteristic, can no longer be so regarded; as, since 1859, when his paper was published, the microscope, at first unaided by the various hardening and staining processes, and latterly with their most valuable help, has been brought into use for the examination of the nervous structures, and the number of fatal cases of acute ascending paralysis in which no pathological change can be found has been proved to be very small. In the great majority there are microscopical changes in the cord and in the meninges, such as could not be detected by the pathologists of 1859, and the opinion of the most recent investigators is that the symptoms are due to an acute intoxication of the nervous system caused by microbic toxins.

Dr. Farquhar Buzzard, after a careful consideration of the results of the examinations, bacteriological and microscopical, of these cases, concludes that it has been shown by investigations on man and the lower animals that the clinical manifestations of Landry's paralysis are the result of either a local

infection, a general pyæmia, or lastly and most frequently, of an acute intoxication. Beyond this stage he has himself advanced the pathology of the disease by his discovery of an organism which he regards as specific and as the cause of the disease, although he is careful to say that he does not, as yet, claim to have in any way proved that this is the only microbe that can produce the fatal toxins.

Assuming, then, on the one hand, that it has been proved by competent investigators that Landry's paralysis is an intoxication of the nervous centres, due to infection by specific microbes, and on the other that urinary paraplegia is the result of the spreading from the urinary organs to the lumbar portion of the cord of a specific inflammation, I think we have grounds to justify a probable explanation of the pathology of the cases to which I am directing your attention.

In every infection three factors are necessary—first, the presence of microbes having active infective powers; second, a medium in which these microbes when conveyed to it can develop; and third, the resistant power of the tissues must be weakened. I venture to suggest as a provisional view, that in my cases these factors existed; first, the specific microbes existed in the urinary organs, probably in the bladder; second, at a certain stage of the disease they were conveyed along the same course, almost certainly the nerves, by which inflammation spreads from the urinary organs in paraplegia urinaria, to the cord, in which medium they developed rapidly, producing a virulent intoxication; third, the tissues were weakened by the long-standing cystitis.

Whether this view be accepted or not, whether it be confirmed or disproved by future investigation, the clinical facts remain the same; and although in neither of my cases did my being able to come to a correct diagnosis and prognosis prolong or save the life of the patient, I look forward to the time when the pathological association of this acute ascending paralysis and chronic urinary disease having been definitely established, remedial measures (probably organo-therapeutic) may be discovered which may stop the rapid course of the malady and avert its fatal termination. It is with this hope that I press these observations on the notice of my fellow-practitioners, and especially of those members of the profession who can devote themselves to pathological investigation.

XIII.—*A Case of Accidental Electrocution causing Extensive Gangrene of all the Extremities and Death on the Ninth Day.* By R. CLEMENT LUCAS, B.S. *Read January* 13, 1905.

E. F. was admitted into Guy's Hospital under the care of Mr. R. Clement Lucas on March 4, 1904.

The Report was made by Mr. F. A. Barber.

Family history.—Good. The patient is almost a vegetarian and a teetotaler, and is a strong and well-built lad æt. 15½.

Previous illnesses.—The patient had scarlet fever when a child, and was in hospital for four months with it.

History of the present injury.—The patient was a junior hand at the London Electric Supply Corporation, Blackfriars Road. At this station the electricity is received from Deptford at a high potential pressure and then distributed in smaller amounts to the surrounding districts. The maximum pressure is 10,000 volts. Along the sides of the main room are placed "transformers" or "converters," which possess an iron casing, 4ft. 6in. high, barrel shaped, and with a complete iron top. The transformers are arranged on each side of the room, one side only being "alive" or in use at a time. The patient got up, as was his custom, on the top of one of these that were "dead" or out of use, to dust some horizontal slabs of slate above them. He was standing on the transformer in one corner of the room, and to his right, as he was dusting, were two more horizontal slates fixed to the other wall, the lower one being about on a level with his shoulder. Between the two were the cable terminals of the high-pressure current of 10,000 volts. The current was running at the time. The patient, after dusting, turned to his right to descend, when the iron plates on the soles of his boots slipped on the iron of the transformer and he overbalanced. (The "linesmen" are provided with rubber boots and gloves, but as the patient was never supposed to be near any "live" wires, he was not given any.) Feeling himself falling, he clutched at the horizontal slates, but missed them, and his *right hand* struck the terminals, thus connecting the current with earth through his body and the transformer. Another

man, working at the other end of the room, noticed the flash and "roar" of the escaping current and looked round, and saw the patient hanging rigid between the terminals and the transformer. He ran across and caught the patient as he fell. The current was not passing through him more than 30 seconds. In falling he struck his eye against the slate, and caused much bruising. It is also possible that the left hand struck the terminal as well, but this is not probable. As we shall see later, both hands were burnt. On being laid on the ground he was rigid and apparently lifeless. The limbs were burnt, swollen and stiff. Respiration had ceased, and the pulse could not be felt. *Artificial respiration* was immediately started and the body vigorously rubbed. In *three* minutes the patient had revived, and shortly afterwards a doctor and ambulance arrived and he was sent up to Guy's Hospital.

Condition on admission (10 *a.m.*).—Symptoms of shock were not so marked as might have been expected, considering that two men had been killed by the same amount of current at the same place.* The patient was quite conscious and soon became very restless, the pain of his burns being very great. The heart and lungs were in good condition and comparatively normal. Pulse, 64; temperature, 97; respiration, 24. The burns were confined to the limbs. Both hands and feet were affected and the right arm up to the axilla.

The *right arm* had a curious appearance—the elbow was semi-, and the fingers completely flexed. The forearm and hand were semi-supinated with the palm directed upwards towards the face. From a line about 3 inches above the elbow down to the extremity, the whole limb was swollen and in a condition of *solid œdema*. It was absolutely senseless and paralysed, and no pulse could be felt.

The pulse had to be taken from the facial artery as it could not be felt at either wrist or ankle.

Just below the axilla was a constricted zone where the skin was adherent and tightly contracted, but not burnt. The rest of the upper arm was swollen, the skin was coming off, and, where it had already done so, the surface was (after hot fomentations) a vivid scarlet. Below the elbow the arm was purple in places, while the hand was a dead white colour. The *skin* seems to have been blown off the limbs, and the joints of the knuckles of the index, middle, and ring fingers of the right hand were exposed and looked white and glistening.

The *left hand* was the least hurt of the four limbs. It was

* *Vide* note at end.

not paralysed, and sensation had not been lost. It was superficially burnt on both dorsal and palmar surfaces and up the forearm for about three inches.

Both feet were very badly burnt, and there was anæsthesia and paralysis up to a point about four inches above the ankle-joints. The soles of the feet were deeply scarred and burnt (and later examination showed that all the tissues down to the ligaments were burnt, several tendons being divided. The ligaments themselves were unharmed).

The tissues here, as in the right arm, were in a condition of solid œdema and were purple and white in colour.

Most of the cuticle was hanging loose or was peeling off, and the numerous bullæ were soon burst by the hot boracic fomentations which were at once applied.

There was a slight burn in front of the left axilla, and it was suggested that this might be due to the metal clip of the braces.

The socks were completely burnt, also the inside of the boots. One boot was burst through at the instep, and the toe-caps seemed to have been blown off. The other had several small holes burnt through it, but on the whole the outside was not affected nearly as much as the inside. The rest of the clothes were not affected, nor (I believe) were the coins and keys in the pockets. The metal in the boots had not been fused.

March 4, *continued.*—By 2 o'clock p.m. (he entered the hospital at 10 a.m.) the *temperature* had fallen to 95°, and *pulse* had risen to 130.

March 5.—Hot fomentations are applied four hourly to all four limbs. Mr. Lane saw the patient and thought it best to wait, before deciding on amputation, until a good line of demarcation should appear.

March 6.—Sensation seems to be returning here and there on the feet, but not in the toes or plantar surfaces.

March 7.—To-day coloured sketches were made of the limbs. In the afternoon Mr. Lucas saw the patient, and decided to amputate the right arm, which had become very gangrenous. This was successfully performed at the shoulder joint and there was very little bleeding. There was a momentary cessation of breathing when the nerves were cut across. The deltoid flap was sewn to the skin of the body by salmon-gut sutures. The amputated arm was *examined*, and showed a condition of solid œdema right up to the axilla. The hand and lower three inches of the forearm were in a

condition of dry gangrene, and from this point upwards there was moist gangrene. The *artery* was normal down to the superior profunda; here it was tightly constricted and thrombosed. The whole of the ulnar artery and superficial palmar arch were thrombosed, the radial artery only slightly. The *veins* contained fluid blood.

March 7, *continued.*—The two feet are dressed every two hours as they are gangrenous. The left hand is dressed with boracic ointment.

March 8.—The patient has made a very rapid recovery from the effects of the operation. He was not sick and was able to sit up in bed without help for his dinner to-day.

March 9.—The wound looks in very good condition, and shows every sign of healing up well. *Temperature* rose to 101·4 last night, but the patient seems very well. He is, however, very restless. The right foot looks bad, and the recovery of either foot is considered unlikely. Creolin fomentations are now being applied four-hourly.

March 10.—This afternoon it was seen that it would be unsafe to delay amputation of the feet any longer, as the gangrene was spreading upwards, and the patient's general condition failing. The patient was therefore anæsthetised, and a double amputation was performed as rapidly as possible. The bones were sawn through, one about half-way down the shin, the other at the seat of election, the operations being simultaneous. At the same time a saline infusion of 12 ozs. was gradually injected into the left axilla. The skin flaps were lateral. In the left leg a condition of œdema could be seen extending up round the muscles almost to the knee. Very little blood was lost. The skin flaps were sewn up with interrupted salmon-gut. Gauze drains were left in posteriorly. The patient bore the operation very well, but was so restless next night that he pulled one leg out of both splint and dressings. The gauze, however, remained on.

March 11.—This morning the shoulder and both legs were dressed under an anæsthetic. The shoulder looked in splendid condition, but, on removing the gauze drains from the legs, a quantity of blood-stained serum gushed out. Gauze drains were again put in.

March 12.—7. 30 a.m.: *Temperature,* 104·2°; *pulse,* 128; *respiration,* 40. The patient's mind is wandering, and he shows symptoms of toxic poisoning. *Later*: The two legs were dressed again to-day, and as both wounds were tensely filled with fluid, nearly all the stitches were removed. The

patient looks very ill. 11.30 p.m.: *Temperature* has again reached 104°. *Pulse* is a running one, and the *respirations* are shallow and very rapid, nearly 1 per second.

March 13.—The patient died at 12.30 this morning.

Additional Notes.

Two men were killed at the same station about eight years ago by the 10,000-volt current. On this occasion a man was working near the 2500-volt terminals and happened to touch them. He was shot away, and came in contact with the 10,000-volt terminals. One was touching the nape of the neck and the other the small of the back. He was rendered rigid and unable to get away. A friend went to his assistance and caught him under the armpits. He was likewise rendered incapable of moving, and as the current could not be turned off at the station, it was passing through them for some time—in fact, until the accident had been telephoned down to Deptford and the current turned off there. They were then quite dead.

Another man at the station touched the 2500-volt terminals and was thrown fifteen feet across the room. He recovered, however, but his arm was paralysed for several days. Another case of complete recovery from a shock of 2500 volts occurred about a year ago at the British Thomson Houston Works, Rugby.

One of the linesmen on the Blackfriars station vouches for a case of a man killed by a 90-volt current. The man who was killed was, however, a heavy driuker. The killing power of the current seems to depend not so much on the actual voltage as on its character (alternating or continuous, etc.), and its volume.

A current with a potential of 17,000 volts may with safety be passed through a person. It has no effect whatever on the body or clothes.

Letter from the Engineer of the Station.

The London Electric Supply Corporation, Limited,
234A, Blackfriars Road,
London, S.E., March 30th, 1904.

Dear Sir,—In reply to yours of yesterdate, I should approximate the amount of current that passed through the body at five ampères. I found by experiment, some years

back, that the ohmic resistance of the human body (in life) from hands to feet varied from 2000 to 6000 ohms, according to the nature of the tissue, the drier tissue offering the highest resistance.

The lad Furlonger was of a moist nature, so I give his resistance at the minimum of 2000 ohms. Therefore, as according to Ohm's law, the volume in ampères passing through a body equals:

$\frac{\text{E. volts or pressure}}{\text{R. ohms or resistance}}$ we get $\frac{10{,}000 \text{ volts}}{2000 \text{ ohms}} = 5$ ampères volume,

and I should say that, taking the whole of the circumstances into consideration, the above approximation is nearly correct.

Hoping this will meet your requirements,

I remain,

Yours faithfully,

J. H. Baker.

P.S.—According to observations I have made of several cases of electric burning, I am of an opinion that the greatest burning effect locally occurs with large volume and small pressure, but that the greatest damage is done to the organs generally in the case of high pressure and small volume.

I hope this is clear.—J. H. B.

Remarks by Mr. Lucas.—The extensive and rapidly increasing use of electricity for motive power and lighting purposes has greatly augmented the number of deaths caused by electricity during the last few years. In *The Times* of August 23, 1904, no less than four deaths due to this cause were reported. Three of these occurred through crossing "live" rails on railways where electricity is partly used as motive power—one on the Lancashire and Yorkshire Railway and two on the North Eastern Railway, whilst the fourth case occurred from chiselling open the cast-iron pipe in which the Metropolitan Electric Supply Company's Main was carried. In all these four cases death was instantaneous. Two other cases which occurred at the same electric station as the case now reported were also instantaneous.

In the foregoing case the patient under artificial respiration recovered from the immediate result of the shock, and died subsequently from the effects of acute instantaneously produced gangrene of all his extremities. The arm which actually came in contact with the main became gangrenous up to the axilla—the appearance produced being that of very severe burning.

Dissection after amputation showed that the gangrene was

due to plugging of the main arteries. In spite of the terrible injuries sustained by this boy, his vigorous constitution gave us hope on the third day that if the gangrenous parts could be detached he might possibly recover. The right arm was, therefore, first amputated at the shoulder-joint, skin from the back being used to cover the raw surface left after removal of the arm and the skin on the trunk side of the axilla. He reacted well, and at first seemed much benefited by the loss of the upper extremity; but the gangrene of the lower extremities began to extend, and necessitated the amputation of these through the legs before he had sufficiently recovered from the shock of the first amputation. These were amputated simultaneously three days later, and he lived three days longer before succumbing to septic exhaustion. The right arm, which came into contact with the main, suffered most, and both feet and ankles were also rendered completely gangrenous. The left hand suffered far less, and there the injury appeared to be superficial and recoverable. A curious everted wound was found in front of the left axilla as if a force from within had escaped at this point, tearing the skin like an escaping bullet. It was suggested that perhaps this was due to the attraction of the metal of his brace, and that the electric force being here divided the left extremity (except the hand) escaped injury.

The drawings accompanying this paper were made by Mr. R. Wingent.

NOTE.—*Communicated by the Hon. Secretaries from "Electropathology. Injuries from Lightning Strokes and Electrical Currents of High Tension." (Electropathologie. Die erkrankungen durch blitzschlag und electrischen starkstrom.)* By DR. S. JELLINCK.

Electrical currents are harmful according to the tension of the current and the resistance of the human body. A tension of 100 volts demands precautions, a tension of 200 volts or more may be dangerous, a tension of 500 volts may be fatal. But the harmful character of the tension is affected by the resistance. A man was killed near Prague

PLATE I.

Illustrating Mr. R. Clement Lucas's paper on Accidental Electrocution.

Upper extremity and one foot, showing extensive burns, the result of the electric discharge. The knuckles and fingers were denuded of skin, leaving the bones and ligaments exposed. On the arm were yellowish patches of dry gangrene and mottling, and on the arm a red blush of inflammation. Gangrene is also shown on the foot, where a more dusky mottling occurred.

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by a current of 95 volts, when standing barefoot in a mixture of potash and sugar. On the other hand, an electrical employee in Vienna, though much damaged, was not killed by a current of 5500 volts. Under "resistance" there is to be considered the resistance of entry and the resistance of exit. As regards the resistance of entry, there is the condition of the clothes, dry or wet, or of the skin. The dry, horny palm of a workman may present a resistance of as much as 100,000 ohms; soft, moist skin, also lesions of the skin, have a very much diminished resistance, whilst the resistance of the mucous membrane of the mouth falls below 1000 ohms. The resistance of exit depends upon the nature of the ground, also, as it is usually the feet which connect with the ground, the covering of the feet. An electrical employee may come in contact with currents at high tension, and do so with impunity, if the feet are covered with non-conducting material. In the case of the workman above mentioned, standing barefoot in potash and sugar, the resistance of exit was very small indeed. The condition of the ground is most important; in private houses, and some mills, the ground forms a good protection; mineral acid factories and coal-mines, on the other hand, especially favour the occurrence of an accident.

In making rules for the prevention of accidents, rooms and shops containing electrical installations should be distinguished, according to the tension of the current and the nature of the ground, into "the safe" and "the dangerous."

XIV.—*A Case of "Soft-Valve" Mitral Stenosis in which no Murmur was heard.* By WILLIAM EWART, M.D., F.R.C.P. *Read February* 10, 1905.

THIS case is of clinical interest in connection with the absence of murmurs; but its importance lies in the association of this fact with a special type of lesion. The variety of mitral stenosis displayed in the specimen before the Society is probably not uncommon, much less rare; but its separate description, if it has ever been written, has not found a prominent place in clinical or pathological literature.

It may at once be stated that absence of induration is the distinctive feature of the type in question in its pure form, which is displayed in this instance. And it has seemed to me that without burdening our classification with any unnecessary additions the term "soft-valve type" of mitral stenosis might be used, at any rate for the purposes of this communication, as a general term applicable also to other less perfect instances and to the whole group, and that it might help to call attention to the clinical and pathological aspects of a study of this condition.

The patient, M. H.,* a woman, æt. 43, was admitted in March, 1904, as a case of "mitral and tricuspid regurgitation with cardiac dilatation," a diagnosis which was maintained until she died, in May. The anasarca of six weeks' standing was general, but much greater in the left breast, side, and limbs than elsewhere, partly owing to the left-sided decubitus habitually assumed since the initial attack of pleurobronchitis in December, 1903.

Fifteen years ago she had had rheumatic fever, with heart affection, but had not suffered in the interval, except from slight palpitation on running upstairs; and as a child she had been fairly active, though inclined to be stout. On admission she was dyspnœic, flushed, and dusky, with large heart and liver, a small, uneven, and irregular pulse (96 per minute), not quite free from tension, and 36 respirations per minute. The heart's action was irregular, the first sound short, the second pulmonary sound relatively loud and rather accentuated. There was no murmur; this fact, independently noted by the

* *St. George's Hospital Medical Register* No. 435; *Post mortem and Case Book* No. 167, 1904.

registrar, Dr. Vere Pearson, and by myself, is repeatedly mentioned in the clinical notes taken at St. George's Hospital, where she gradually improved under treatment. She was transferred to the Wimbledon Convalescent Hospital, where she relapsed and died. One of the notable features of the case was the profuse spontaneous perspirations which contributed to her temporary improvement. The existence of mitral stenosis was never suspected by myself or by other observers.

The heart was described by Dr. Salusbury Trevor as slightly enlarged as a whole (weight 18 ozs.). It presented definite though not considerable dilatation of the right cavities. The left auricle was moderately dilated; and the left ventricle slightly dilated also. The heart's substance was rather soft and sodden, and slightly fatty.

The pulmonary and aortic valves were competent (by test). The tricuspid orifice was slightly enlarged, and the edges of the valve slightly thickened. Alone the mitral valve was profoundly altered, its stenosed orifice admitting only the tip of an average finger. The shallow funnel formed by the united flaps was of the usual shape, but in consistence entirely different from the usual type. The valve membrane was neither very thick nor stiffened by hard fibrous tissue or calcareous deposit. Its pliability was equal to that of soft indiarubber. Still more striking was the fact, well displayed in the specimen, that the chordæ tendineæ were free from thickening and shortening, and that the musculi papillares were quite normal in appearance.

Remarks.

Absence of murmur in fatal degrees of loss of compensation is not uncommon in dilatation of the right heart with tricuspid regurgitation, a diagnosis which was attached to the present case for want of any other indication. Although it is well known that in the terminal stage of mitral stenosis the characteristic murmur may escape detection, whilst in the early stages there is often a tendency to its temporary abeyance, as a fact a bruit does not fail to reveal itself sooner or later when the case is under continuous observation, particularly when, as in this instance, the heart has been examined by various independent observers, not only during cardiac prostration but during intervals of improvement. Indeed, in my experience it is less common that a murmur should be permanently absent and an existing stenosis overlooked than

that stenosis should be diagnosed in error because a murmur was heard which closely resembled the characteristic bruit.

In this instance a murmur may have been audible at some previous time, but during the last eight weeks of the patient's life it was never heard either by myself, my house-physician and students, or the medical registrar—all working independently—nor by the visiting physician and resident medical officer at the Wimbledon Convalescent Hospital. I believe this to be an unusual experience in mitral stenosis, and in itself to be worthy of record.

An explanation for this anomaly may perhaps be contained in the anatomical peculiarity to which reference has been made. At any rate, there is a possibility that this case might be of use, together with other future cases, in contributing a new and helpful factor to the desired elucidation, namely, a clearer recognition of the separate type of lesion which it so well illustrates.

The most recent classical descriptions of the lesions of mitral stenosis present very little variety; they are confined to the terminal lesions of the heart, and usually touch little, if at all, upon the antecedent conditions. A distinction is, however, made between the two extreme types commonly described, to the effect that the "funnel" variety is more prevalent in the juvenile group of cases and the "button-hole" in the adult. The former group is clinically remarkable for the long duration of the lesion and the repeated history of loss and restored compensation. The "funnel" must be admitted to be always a much advanced lesion, corresponding always, I believe, to a typical murmur. Nothing resembling that formation belongs to the earliest stages of the affection; and the fact is significant that those earlier days are those when an inconstancy of the mitral stenotic murmurs is often noted. From this two inferences may perhaps be drawn: (1) that the more advanced type is that most apt to produce the murmur; and (2) that the button-hole variety, or variety without a "funnel," may perhaps be regarded as a less complete and earlier stage of the disease, less apt to lead to a characteristic murmur.

To return to the specimen under discussion, it differs from both extreme forms in being free, not only from much induration of the valve, but particularly from any implication of the chordæ tendineæ and of the musculi papillares, the result being a nearer approach to the "button-hole" type than to the "funnel." It really represents a separate third variety

of mitral stenosis, the "soft-valve" type, where the lesion may, as in this case, or may not be strictly limited to the flaps of the valve. This soft variety might perhaps be found, if looked for, in the hearts of chronic mitral cases ending prematurely through accident; for it is clear that the late stiffening and calcification must be preceded by stages of moderate thickening.

As regards the mode of production of the murmur of mitral stenosis, this case and specimen lend definite support to the view, which is largely entertained, that the bruit is connected with the resistance offered by thickened and rough chordæ tendineæ to the direct current from the auricle.

XV.—*A Case of Accessory Thyroid laterally placed in the Floor of the Mouth.* By E. Percy Paton, M.D., M.S., F.R.C.S. *Read February* 10, 1905.

M. S., a girl, æt. 15, was sent to Westminster Hospital in April, 1903, on account of a swelling in the floor of the mouth.

She had noticed the lump for about four months, and thought that it was increasing in size a little; but except for the feeling of a swelling it gave her no other inconvenience.

On examination a mass could be easily felt on the right side of the floor of the mouth, which seemed to be about the size of an ordinary walnut, with the green skin removed. Its situation was well to one side of the middle line, and it did not extend quite up to the frænum linguæ. It could not only be felt in the mouth, but also in the right submaxillary region, and its outline was best made out with one finger inside and the other outside the mouth. In consistency it was firmly elastic, and I was uncertain if it contained fluid or not. It did not lie immediately under the mucous membrane, but appeared to lie beneath the sublingual salivary gland. It did not seem to be at all freely movable in the tissues among which it was placed, but owing to its situation this point could not be clearly determined. No definite diagnosis was made before the operation, but it was thought that it might possibly be a cyst, or more probably a solid growth connected with one of the salivary glands.

As it did not seem that the mass could be easily reached through the mouth, an incision was made over it in the neck, and after cutting through the mylohyoid muscle the swelling was fully exposed and was found to lie, as had been expected, beneath the sublingual gland; it was easily removed, though it did not shell out well, but had to be freed by cutting from the condensed tissues around it. This tissue was more vascular than one expected; there was no opening made through the floor of the mouth, and the wound healed without event.

The swelling when removed was about the size of a walnut; unfortunately, the main mass was accidentally thrown away after microscopic sections had been made, so that I am not able to show it to-night, but the microscopic section which is shown demonstrates the structure to be that of ordinary thyroid tissue, with in some places some dilatation of the spaces

filled with colloid material, clearly showing the mass to be an accessory thyroid gland. A fairly defined capsule surrounds the glandular tissue.

Accessory masses of thyroid tissue are not very uncommon in various parts of the what has been called the "thyroid tract"—that is to say, in a region extending to a variable distance on either side of the middle line, and reaching from the root of the tongue about the foramen cæcum to the isthmus of the thyroid body; but I have not been successful in finding a case recorded in which such a mass has been placed in the floor of the mouth well to one side of the middle line as in my patient.

The various seats in which accessory thyroids have been described as occurring are classified as (1) below, (2) behind, (3) laterally, (4) above, and (5) in front of the normal gland, and all the laterally placed ones seem so far to have been described as occurring in the neck. Those classified as occurring above the normal gland have been placed in the middle line about the body of the hyoid bone, in the floor of the mouth, below the frænum of the tongue, in the middle of the tongue itself, or at the root of the tongue in the middle line, forming a swelling in this situation, pushing the mucous membrane in front of it, and perhaps pressing down the epiglottis.

The patient's attention has been called to the trouble sometimes, as in my case, merely by the existence of the swelling, in other cases by difficulty of swallowing, interference with respiration, snoring, or alteration of the voice; occasionally there has been a sudden increase of size in the mass due to hæmorrhage into its substance, or again in other cases the first trouble has been bleeding into the mouth, which may have been severe, and coming from the large vessels overlying the tumour between it and the mucous membrane. These vessels may be so large as to be a serious consideration in removal of the mass. In one case described by Seldowitsch the thyroid in the root of the tongue was the only mass of this tissue the patient possessed, and its removal was followed by myxœdema; this led to an examination of the neck, and no thyroid could there be found.

The mass may be noticed at any period of life from infancy to old age, but in most cases the swelling seems to come under notice and to increase in size between the ages of 15 and 25. They occur more commonly in women than in men; in one series of cases there were twenty-eight men and forty-nine

women. The diagnosis is often uncertain before operation, and lies between the various forms of cyst that occur in this region, enlarged lymphatic glands, tuberculous or otherwise, some form of simple growth, sarcoma, carcinoma, gumma, actinomycosis, or angioma; no doubt in the majority of cases the nature of the mass can only be suspected before it is removed. In cases in which a growth is suspected to consist of thyroid tissue, it is well to remember that there may be a good deal of bleeding during its removal, and this should be provided for by a free exposure of the mass; if necessary, the jaw may be divided or the cheek split, and in cases involving the base of the tongue a preliminary tracheotomy may be a wise precaution.

A very full account concerning accessory masses of thyroid tissue occurring in the mouth, by Chamisso de Boncourt, will be found in the 'Beiträge zur Kliniche Chirurgirie,' vol. xix, 1897.

XVI.—*Extra-Peritoneal Uretero-Lithotomy in a Child aged three years.* By Henry Betham Robinson, M.S.Lond., F.R.C.S. *Read February* 10, 1905.

On two previous occasions I have elsewhere recorded successful cases of extra-peritoneal uretero-lithotomy occurring in adults. Reference to other published cases shows no instance where this operation has been previously performed at such an early age, and this fact alone, apart from the desirability of recording all these cases, makes this case worthy of notice.

On June 17, 1904, G. C., a male child, æt. 3, was admitted to the East London Hospital for Children, Shadwell. On the 16th he had pain in the penis, worse on running about, and in the evening he passed small quantities of urine, frequently with great penile pain, but no blood, which the mother looked out for. She gave him oil, and the straining to defæcate made him much worse. He was admitted to hospital mid-day on the 17th, not having passed urine since 3 a.m.

Examination showed an impacted calculus in the bulb, with the bladder distended up to the umbilicus. Very shortly afterwards this stone was cut down on and removed with a scoop, and there was a free flow of clear urine. The wound in the urethra was closed with fine silk, and the skin wound only partially closed, the remainder being lightly packed with gauze. Before closing the urethral wound a sound was passed into the bladder, but no other stone was detected. A bimanual examination confirmed this and gave us some more information, for a small hard lump was felt on the left side of the pelvis just below its brim, which I diagnosed as an impacted calculus in the ureter; the kidney did not appear to be enlarged. Not knowing how long the calculus had been here, and supposing that perhaps it was making its way to the bladder, I determined to watch it for a few days.

The urethral wound healed without any leakage, and on examination at the end of a week the ureteral calculus remained in the same position; I assumed it was fixed and arranged to remove it.

The urethral calculus was about the size and shape of a cherry-stone, measuring $\frac{7}{16}$ by $\frac{3}{16}$ in. and showing no facets.

The operation of uretero-lithotomy was performed on the 28th. A curved incision was made on the left side as if to

tie the external iliac artery, and the peritoneum was pushed upwards and inwards till the inner side of the vessels was reached. The little hard lump in the ureter was felt just below the brim of the pelvis, and the delicate fat was separated to expose the ureter. A finger in the rectum enabled the ureter to be pushed well up into the wound; a small longitudinal incision was made down on to the stone, which was lifted out with a small scoop. There was an immediate gush of pale odourless urine, estimated at about two ounces; so that, although no enlargement of the kidney could be made out, there must have been considerable distension of its pelvis and calices. A fine catgut stitch was put through the outer coats of the ureter, avoiding the mucous membrane, and a small drainage-tube with a gauze wick inside was placed in close relation with the ureteral wound. The muscles and skin were sewn up together with horsehair.

The calculus was of oval shape, slightly flattened, and roughened on its outer side with a coating of oxalates; it measured just under $\frac{3}{16}$ of an inch in length, and $\frac{2}{16}$ at its thickest diameter. A section shows it is composed of alternating laminæ of uric acid and oxalates.

On the 29th the wound was dressed and the gauze wick removed and renewed. There had been some discharge of urine into the dressings. The child's condition was most satisfactory. The drainage-tube was taken out on the third day, the gauze being only a little damp, but a piece of gauze was replaced between the lips of the wound. After the fourth day there was no suspicion of any leakage of urine. The child's state was perfect, a normal quantity of urine being passed and at regular intervals; there had been no blood in the urine since the operation. There was never any rise of temperature after the operation. The wound was quite healed on July 13.

The experience of my three cases enables me to state that it is a far easier operation in the child than in the adult, for the following reasons, viz. the thickness of the soft parts is less, the peritoneum strips back more readily owing to the small amount and delicate nature of the subperitoneal connective tissue (although great care must be taken not to wound the very thin peritoneum), and the small size of the pelvis with the abdominal position of the bladder enables the finger in the rectum to push the ureter with the impacted stone well up into the wound instead of having to work at the bottom of a deep space.

One point brought out in this case I would wish to emphasise, viz. that with an impacted urethral calculus it should not be assumed that this is the only one. Examination of the bladder with sound and bimanually as well as palpation of the abdomen may discover a second or more stones which, if left behind, may produce serious results; in this case especially the unsuspected impacted ureteral stone would assuredly have caused hydronephrosis if left in situ.

On the technique of the operation there is nothing to comment. It was extra-peritoneal, as is now firmly established it should be. The lips of the ureteral wound were brought together with catgut, which did not go through the whole thickness of its wall; if possible to do this, it is better, as it limits, if not prevents, the discharge of urine and induces rapid healing. Of course drainage down to the ureter must be still used to circumvent any urinary extravasation.

XVII.—*Calculous Anuria in a Child.* By Douglas Drew. *Read February* 10, 1905.

S. T., æt. 4. Admitted under my care at the North-Eastern Hospital for Children on April 27, 1904, with stone in the bladder and cystitis.

On May 3 suprapubic lithotomy was performed. The bladder was found to be greatly hypertrophied. It was drained by a tube, the wound being closed around it. The urine continued to drain from the wound until May 22.

On May 27, owing to the child having had some pain in the abdomen for two days, and an elevated temperature, the wound was opened up in order to allow of irrigation and further drainage of the bladder, as the urine was offensive and contained pus.

On June 7, as the temperature had remained elevated since previous note, the bladder was examined under chloroform, drainage-tube re-inserted, and irrigation continued.

After this the condition improved until June 24, when the temperature again went up, and the child was sick.

On June 28 there appeared to be some tenderness on palpation over the left kidney, and this, with the irregular temperature and the persistence of the pyrexia, suggested a secondary nephritis.

On July 2 the temperature had become normal, and the child was much better.

On July 19 the leakage from the wound had almost ceased, and the urine contained a few pus-cells and was acid.

On July 24 again complained of pain in the abdomen. The temperature again rose irregularly, and remained up until August 10. During this time the urine was acid, and contained some mucus and a little pus.

From this date until August 28 the temperature was normal and the sinus above the pubis practically healed. The average quantity of urine was 25 oz. On this date the child was seized with severe pain, and a small calculus was found impacted in the meatus and removed under chloroform.

During the month of September the child improved rapidly. The wound remained healed, the temperature was normal, and the urine was acid, and contained only a little mucus. The child was allowed to get up and play, and was about to be sent home, when he again had pain in the region

of the scar, and on September 28 some leakage above the pubis occurred.

On October 1 the child was sick and again had pain in abdomen.

On October 4 the urine was alkaline, with blood and pus. X-ray of bladder and kidneys negative. The wound continued to leak, so that it was difficult to estimate the total amount of urine; the quantity passed *per urethram* varied between 7 and 19 oz. The quantity was also doubtless diminished, owing to the vomiting which occurred several times daily.

October 9 and 10 2½ oz. were passed *per urethram.*

On October 10 the child had another attack of pain, referred to the left inguinal region. Another X-ray was taken, and showed a doubtful shadow about the brim of the pelvis on the left side.

October 11. Examination under chloroform. The left kidney was felt to be enlarged; the right kidney could also be felt, but did not appear enlarged.

Per rectum a stone was felt in the left ureter, just below the brim of the pelvis.

Operation, October 11.—The left kidney was at once exposed from the loin, and after dislocating it on to the loin, it was incised, and a calculus found in the pelvis, and removed. The ureter, which was much enlarged, was then traced downwards, after extending the incision forwards until the stone was reached, about two inches above the bladder. It was gently worked upwards along the enlarged ureter until it was within an inch of the pelvis, when it was removed by incising the ureter. The kidney was replaced, but before closing the wound the finger was passed across the abdomen outside the peritoneum in order, if possible, to palpate the right ureter. It could be readily felt lying on the psoas, and was much enlarged. The left kidney was drained and a second tube inserted down to the ureter.

If the child's condition would have permitted of further interference, I should have at once explored the right kidney, as from the size of the ureter I was convinced that there was mischief on the right side, although I had overlooked the stone which *post mortem* was found in the lower end of the ureter.

After the operation the child suffered severely from shock. The vomiting persisted. The child was drowsy, and towards the end muscular twitchings appeared. The quantity of urine passed *per urethram* gradually diminished from 8 oz. to 2 oz.

on the fourth day. Saline injections *per rectum* and intravenous were given, and hot-air baths, but the child's condition did not improve sufficiently to permit of the exploring the right kidney, and for three days before his death only 2½ oz. of urine were passed *per urethram*. A considerable quantity drained from the left kidney, so that there was not total anuria, as the title of the paper indicates.

Remarks on the Case.

Inasmuch as failures are often more instructive than successes, I offer no apology for bringing an unsuccessful case before the Society. In reflecting upon it—and it is easy to be wise after the event—I have two regrets: the first that I did not make an examination of the rectum at an earlier date, for even supposing that I had overlooked the stone in the right ureter, as I did at a later date, I should have been able to have removed the stone in the left ureter before the onset of the urgent symptoms.

The other regret is that, when I found the right ureter enlarged at the time of operation, I did not take the risk and explore the right kidney, although the child's condition was very unfavourable, the shock being so severe. Throughout its course this case was remarkably perplexing.

The persistence of the pyuria after the bladder had been drained and irrigated regularly for some weeks, together with the irregular attacks of temperature, suggested a consecutive nephritis, following on the cystitis.

The very slight amount of abdominal pain, with almost complete absence of tenderness over the kidneys, seemed to exclude the possibility of stone in the kidney, and rendered the probability greater that the trouble was due to some infection of the left kidney, over which there appeared to be a slight degree of tenderness at intervals.

After the small calculus was removed from the urethra on August 28th the improvement was so remarkable that I was forced to think that all the symptoms had been occasioned by the irritation of the stone. The temperature kept normal, the sinus above the pubis was firmly healed, the urine was acid, and contained only a little mucus and pus. In fact, we were on the point of discharging the child when he began to show signs of further trouble. It was not, however, until October 9 that the pain became severe, and was referred to the left inguinal region. The radiograph taken on October 6

was negative, and the second did not materially assist us, beyond that there was a doubtful shadow on the left side of the pelvis.

From the marked degree of hypertrophy and dilatation of the ureters, there is no doubt that both the stones in the ureters had been there for a long time. To my mind the extraordinary feature of the case is that these stones in the ureters should have caused so very little pain. It is true that the child appeared to dislike the abdomen being palpated, and on one or two occasions there was tenderness over the left kidney, but it was not enough to cause him to cry, and there was never anything resembling an attack of renal colic until October 10, and so far as the right side was concerned all symptoms were entirely absent.

I wish to emphasise this point, as the absence of pain and tenderness was the chief feature of another case which I brought before the Society for the Study of Disease in Children, and in which I removed sixty-seven small calculi; the chief symptom in this case was hæmaturia.

It was extremely mortifying to find that I had overlooked the presence of the stone in the right ureter, as from its situation, immediately above the entrance into the bladder, it must have been quite easy to feel; the absence of all symptoms from the right side accounted for this mistake, such localising symptoms as there were directed attention only to the left side.

The first positive sign I found of trouble on the right side was at the time of the operation, when I could distinctly feel the right ureter to be enlarged on palpating it on the psoas muscle, across the abdomen, through the wound.

General Remarks.

Stone in the kidney in childhood is a rare affection. I have already referred to a case of nephrolithotomy in a child I published in the *Transactions* of the Society for the Study of Disease in Children, 1904. This case, together with the one under notice, and one that was operated upon by Mr. Bilton Pollard, are all that are to be found in the records of the North-Eastern Hospital since the introduction of the operation in 1880.

Other surgeons with similar experience in children remark on the rarity (Ashby and Wright, three cases).

The case is extraordinary in that calculi were removed from the urethra, bladder, ureter, and kidney.

In dealing with stones that have been located in the ureter two chief methods are open to selection: (1) To cut down and remove the stone, *i.e.* to perform a formal operation of uretero-lithotomy; (2) to explore the kidney and ureter through the oblique lumbar incision, extending the incision forwards into the inguinal region, as the occasion may demand, in order to remove the stone from the ureter. Before deciding which of these courses shall be adopted, certain important points have to be determined.

(1) *The position of the stone.*—If it is situated at or near the entrance to the bladder, some special method for its removal is indicated, such as removal through the bladder or by the sacral or vaginal route. Whether this shall be done at once will be influenced by the condition of the patient and of the kidney above. If anuria is present, owing to disease or absence of the kidney on the opposite side, it is imperative to relieve the kidney by drainage, and to deal with the stone at a later date. The same treatment is indicated when pyonephrosis is present above the stone in the ureter. (2) The presence of septic pyelitis. (3) Positive evidence by radiography of other stones in the kidney of the same side.

If either of these two conditions is present—and in the majority of the cases one or other is frequently found—a formal uretero-lithotomy is clearly contra-indicated. Under these circumstances the kidney should be explored and the stones removed from it, and then the ureter should be traced down to the position of the stone, and the latter removed. The method of removal will depend whether it is impacted or not. If it is not, then it should be gently manipulated upwards along the ureter, and removed by incising the pelvis, as was done in the case under notice, or by extracting it through the incision already made in the kidney. If it is impacted, the ureter must be incised longitudinally in order to remove it.

All cases of impacted stone in the ureter as low as the brim of the pelvis can be reached through the lumbo-inguinal incision.

The pyelitis is remedied by free drainage of the kidney.

The question, therefore, of impaction of the stone in the ureter appears to me to exercise very little influence in deciding upon the procedure to be adopted.

I would summarise the matter in this way: All cases of stone in the ureter as far down as and including the brim of

the pelvis should be dealt with by exploration of the kidney and ureter through the lumbo-inguinal incision, which has the advantage that any stones present in the kidney can be removed, and also that the kidney can be freely drained if pyelitis is present.

Exceptions to this rule may be found when there are clear indications that both pyelitis and other stones in the kidney are absent; under these circumstances, it may be preferable to perform the operation of uretero-lithotomy without exploring and draining the kidney.

With regard to the value of radiography in determining the presence of a stone in the kidney, if there is positive evidence it is satisfactory, but a negative result by no means excludes the possibility of a stone being present. This point should be remembered before deciding upon uretero-lithotomy.

XVIII.—*A Case of Acute Rheumatoid Arthritis.* By W. Hale White. *Read March* 10, 1905.

FROM among the diseases which are often grouped together under the names of chronic rheumatoid arthritis, osteo-arthritis, arthritis deformans, and chronic articular rheumatism, there is one which I would suggest should be sharply separated off as a distinct disease. It has, I believe, the following characteristics: It is usually met with in women whose age is most often near to 20; the disease begins, or is at any rate soon most prominent, in the proximal interphalangeal joints, and is quite early strikingly seen in the wrists; it is markedly symmetrical in the two hands, the swelling is considerable and extends beyond the joints, so that there is a well-marked fusiform swelling about the affected interphalangeal joints and a general swelling about the wrists and other joints; sometimes a creaking may be felt, due, apparently, to thickening of the synovial membrane. In the course of a very few weeks most of the joints in the body are affected, so that the woman is soon bedridden, unable to move fingers, thumbs, wrists, elbows, shoulders, and the corresponding joints of the lower extremity. The joints of the spine are often affected, so that turning in bed is difficult and painful. The temporo-maxillary joint is frequently implicated, and mastication is consequently difficult. Wherever the disposition of surrounding muscles makes it easy to investigate the joints, swelling about them is obvious, and the patient usually complains of pain in them. Muscular wasting is so very rapid that it is clearly an extreme degree of genuine arthritic atrophy and not dependent on disuse; the wasting is especially seen in the hand and forearm, but in a bad case it is almost universal. The temperature is often raised daily, mostly in the evening, to a point usually between 100° and 102° F. The hands and feet are often covered with sweat, while the rest of the body is quite dry. The patient is pale, and the pulse is increased in frequency out of proportion to and apart from pyrexia, although neither from the history nor from the examination of the heart is there any evidence to connect the disease with rheumatic fever. Sometimes a number of brown spots like large, dark freckles appear on the face, body, and limbs.

After two, three, four, or even six weeks, the temperature slowly regains its normal point, the rate of the pulse diminishes,

but more slowly than the temperature. The colour partly returns, the sweating of the hands and feet lessens, the pain passes away, but much immobility and some swelling around the joints remain, and the patient is liable to fresh attacks, which exactly resemble the first, and result in fresh swelling of and around the already fixed joints. After the acute stage has subsided the sufferer is by no means recovered, for the swelling of the synovial membrane and soft tissues around the joint leads to great immobility of them, and much of the muscular atrophy remains; the patient is hence often a chronic invalid. In this respect, as well as in many others, the disease stands in striking contrast to rheumatic fever. Careful manual examination during life, even in cases which have lasted many years, fails to reveal any lipping or bony outgrowths in connection with the joints. The deformity appears to be entirely due to effusion into the joint and swelling of the tissues around. Radiographic examination confirms this view. As a rule the attacks diminish in severity as time goes on, and ultimately the patient may only suffer from the deformities due to previous attacks.

The above is an abstract of the clinical characters of the disease which I wrote in the *Guy's Hospital Reports*, vol. 57, and I have made it rather full, for I want to show that the case I am about to relate illustrates this account of the disease. I should add that in the paper in the *Guy's Reports* I have been fortunate enough to be able to give an account of the histology of the disease and to show that the morbid process consists of a chronic inflammation of the synovial membrane, that there is no inflammation of bone or cartilage, and that any histological change in the cartilage or bone is accidental and due to either being worn away by the pressure of some thick synovial fringe. This accords with the fact that these cases may go on for many years and yet no grating suggestive of erosion of cartilage takes place in any joint.

As this arthritis resembles rheumatic fever in being acute, and in the fact that one acute attack greatly predisposes to others, I would suggest that it be called "acute rheumatoid arthritis," but the account I have just given shows that it is absolutely distinct from rheumatic fever, and its whole course, the absence of bony outgrowths, and the joints affected, show that it is separate from osteoarthritis.

The following is an account of a case recently under my care:

Olive C., æt. 25, was admitted into Guy's Hospital on

March 22, 1904, under the care of Dr. Hale White. She had never had rheumatic fever, chorea, or any disease usually associated with rheumatic fever.

In June, 1903, she complained of pain and swelling in the proximal interphalangeal joints of both index fingers. The pain got better and the swelling decreased, but some of it remained. In September, 1903, she suffered from pains in the lower extremities. Fourteen days after she again noticed pain and swelling of the proximal interphalangeal joints of both hands; afterwards she had pain in the elbows, shoulders, feet, knees, hips, and temporo-maxillary joints. The visible joints were swollen, the pain preceded the swelling, and when it disappeared swelling and stiffness of the joints remained. It is now four months since she was able to open the mouth for more than half an inch. She has been unable to walk for some time, and had to take to her bed eight weeks ago. Also for some weeks past she has been unable to fully extend her knees and elbows, or to bring her right hand to her mouth. She has wasted a great deal, and her hands and feet sweat so profusely that frequently the sweat, to use her own words, "steams off them." She has noticed that lately several small, light brown, pigmented spots have appeared, especially on the face.

On admission.—Her pulse was 148, temperature 100·6°, respiration 24. There was much muscular wasting. Many light brown pigmented spots could be seen like large freckles on the limbs, trunk, and face. Her hands and feet were sweating profusely, although the rest of the body was quite dry. The heart, lungs, abdomen, and urine, appeared perfectly healthy.

Joints.—The condition of the hands was very striking, for there was much muscular atrophy and weakness, and while the terminal interphalangeal joints appeared perfectly healthy, there was considerable uniform spindle-shaped swelling of all the proximal interphalangeal joints. The accompanying photograph gives a better idea of these joints than any words; it shows also the swelling about the wrists, and the muscular atrophy. There was very little affection of the metacarpo-phalangeal joints or of the thumb, but there was much swelling of the wrists and fluctuation could be obtained. There is some swelling of both elbows, which are flexed and cannot be completely extended; both radio-ulnar articulations are affected, both forearms are incompletely pronated, and voluntary and passive movements are limited. Movement in the shoulders and hips is very limited. Both knees are

swollen and flexed to more than a right angle. The ankles are swollen and movement is deficient, and the same is true of several toe-joints, and the swelling is most marked in the proximal phalangeal joints of some of the toes. She can only open her mouth a very little way, and she complains of pain in the spine as well as in several of the joints previously described. In all the affected joints careful examination fails to reveal any lipping or outgrowth of bone, no grafting due to destruction of cartilage can be obtained, and the swelling is clearly due to an excess of synovial fluid, with some thickening of the synovial membrane and the tissues outside it.

During the first week of her stay in the hospital the morning temperature was usually about half a degree below normal, whilst the evening temperature was either between 99° and 100°, or just over 100°; the pulse was never below 100, usually about 120. During the second week the temperature was usually within half a degree of 98·4°, but the rapidity of the pulse remained the same. Whenever we examined her the sweating of the hands and feet was most striking; they were quite wet, whilst the rest of the body was quite dry.

The temperature gradually subsided, and the pain in the joints passed away. The patient was regarded as suffering from an attack of acute rheumatoid arthritis, but the fixation of the joints showed that there had been previous acute attacks, so after the present acute attack had subsided some of the adhesions of the elbows and knees were broken down under gas, and by this and subsequent passive movements and massage considerable improvement in the mobility of the joints has taken place. There are few diseases in which it is more important by passive movements to prevent fixation of joints; if these are not very systematically performed, the fixation becomes very marked in many joints. The difficulty of opening the jaw which usually occurs during an acute attack commonly passes off sooner than does the difficulty of moving other joints, and this is, I think, because the jaw has to be moved when the patient eats or speaks.

The radiographic picture of the hand which I show is, I think, of interest, for I have already mentioned in the paper in the *Guy's Hospital Reports* that the ends of the bones, especially of the hands, have in my two previously published cases been unusually transparent to the Röntgen rays, and this is well seen in the present photograph, the ends of the metacarpal bones and phalanges being strikingly translucent. Future observation alone can show whether this condition is

especially striking in acute rheumatoid arthritis, but other cases of this disease I have seen show this unusual transparency very markedly. Certainly it may be seen to a less extent in other diseases.

It will have been noticed that I suggest that this is a perfectly distinct and separate disease from other diseases of joints. It certainly is not rheumatic fever; the joints usually selected are different, the disease does not flit from joint to joint, it leaves permanent deformities, the sweating is confined to, or at any rate by far most striking, on the hands and feet, the pulse is rapid apart from the temperature, and there is no association with endocarditis or chorea. The fact that it is far more common in women than men shows it is not gonorrhœal, and there has been no evidence of gonorrhœa in any of the cases I have seen. The whole clinical course and the absence of eburnation, and the formation of new bone, show it is not ordinary osteo-arthritis, and I have not noticed any association with tubercular disease elsewhere in the body to suggest that it is tubercular; in my fatal case recorded in the *Guy's Hospital Reports*, vol. lvii, no tubercle bacilli could be found. But the suggestion has been made that it is a chronic streptococcal infective arthritis; thus G. E. Gask (*Path. Soc. Trans.*, vol. liv, p. 319) records the case of a woman in whom a multiple arthritis followed delivery. The clinical description is very meagre, but the case is stated to be a typical example of acute rheumatoid arthritis, and streptococci were found in the fluid removed from the knee during life; but, as Gask says, an isolated case does not prove much, and then, too, it must be remembered that there was no *post-mortem* examination, and he failed to reproduce the disease in either a rabbit or a mouse by injecting some of his streptococcal culture. My fatal case was most carefully examined bacteriologically in the laboratory at Guy's Hospital. Specimens were taken from (1) the blood of the right ventricle; (2) fluid from the right knee; (3) mesenteric gland; (4) spleen; (5) synovial membrane of left knee. Specimens 1, 2, and 4 were sterile. From 3 a small, non-motile bacillus was obtained, which could not be identified, and from 5 a coccus was obtained which was unlike usual cocci and could not be identified either by Washbourn or Pakes. There is, I would submit, at present no bacteriological evidence that the disease is an ordinary streptococcal infection, nor is there any clinical evidence in favour of this view; in fact, I think the clinical evidence is against it, for I have failed to find any source of

PLATE II.

Illustrating Dr. Hale White's Case of Acute Rheumatoid Arthritis.

Shows the spindle-shaped swelling of the proximal interphalangeal joints of Olive C—.

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PLATE III.

Illustrating Dr. Hale White's Case of Acute Rheumatoid Arthritis.

Shows the transparency to the X-rays of the ends of the bones of Olive C—.

Bale and Danielsson, Ltd.

infection in cases I have examined, but there was a typical case of the disease in the hospital a year ago, under my colleague Dr. Newton Pitt, who kindly lets me mention it. The patient had severe pyorrhœa in connection with many decayed teeth. These were removed and the gums, before she left, were quite healthy, but I wrote to her a year after she had left the hospital and learnt from her that she was no better; and, therefore, although the possible source of streptococcal infection had been removed, her joints had not recovered. If such a suggestion may be hazarded, it seems most likely that some day the disease will be shown to be a definite disorder, with its own specific micro-organism.

XIX.—*Arthritis Deformans and its Allies.* By Bertram Abrahams, M.B., B.Sc., F.R.C.P. *Read March* 10, 1905.

I PROPOSE to direct the attention of the Society this evening to the consideration of the clinical features of the various affections which are ordinarily grouped under the name of "rheumatoid arthritis." At the outset I would venture to say that I count myself among those who think that this name should be discarded as both barbarous and unscientific; this point I have already discussed elsewhere,[1] but I refer to it again now, as I think that in this case at any rate it would be well for us to assimilate our nomenclature to that of Continental writers.

At present, however, we have to recognise that the expression "rheumatoid arthritis" is the one in common use in this country, and furthermore that it includes at least four different conditions, the relations of which to one another I propose briefly to analyse. These conditions, for convenience sake, we may arrange chronologically, according to the average age of the patient when first attacked. They are: (*a*) the children's disease described by Dr. Still; (*b*) the acute process occurring in young women, of which Dr. Hale White, who has previously [2] reported two typical cases, has given another example to-night; (*c*) the familiar multi-articular affection of middle-aged women; and (*d*) the equally well-known chronic malady of the aged.

I do not intend to waste the time of the Society by going over details the knowledge of which is common to all, or to relate at any length the history of illustrative cases. I submit, however, a few skiagrams and photographs of some of the more characteristic appearances.

Still's disease.—This is usually referred to as rheumatoid arthritis in children, a most unjustifiable attribution, since in his original paper [3] Dr. Still pointed out with the utmost care how it differed from what had previously been described as rheumatoid arthritis. Although the practice of using personal names in connection with maladies is one generally to be reprehended, I would suggest that until some simple and expressive term be found this should be called "Still's disease." It is undoubtedly an infective process, and differs

from all the other forms which we have under consideration in respect of the enlargement of the spleen and lymphatic glands with which it is almost invariably associated. Dr. Hale White believes that the acute disease which occurs in young women is also infective, and the two are certainly alike in the presence of irregular fever and the absence of "nervous" symptoms, with the exception of considerable muscular atrophy. But in other respects they differ widely; the one is essentially chronic, is more prevalent in boys, and attacks the fingers very late; the other is acute, has chiefly been described in girls, and usually commences in the fingers. Pathologically each is mainly a synovitis, with only secondary changes in the cartilages, but it is only in the juvenile malady that we find enlargement of the spleen, lymphatic glands, and sometimes liver. The condition of the joints in the latter has been carefully studied by Whitman [4]. On consideration of all the evidence, I would regard Still's disease as a chronic specific infection, and would separate it entirely from all other forms of joint disorder.

Senile osteo-arthritis.—The complaint which occurs at the other extreme of the scale of life has equally distinctive characteristics. It is primarily a degenerative process, consisting in fibrillation, breaking up and wearing away of the joint cartilage. There may be some hypertrophy of the synovial folds at the edges, but this is secondary, and might almost be called compensatory. It has indeed been stated (Moll [5], Reyher [6]) that similar pathological changes may result from prolonged immobilisation of a joint. The affection is so little inflammatory that it is doubtful whether the name "osteo-arthritis" should with propriety be applied to it. Further distinctive features are the absence of anæmia and of "nervous" symptoms, and the fact that it usually attacks the large joints, especially the hip, first. It is worthy of remark that in all the other three forms which we are considering, as also in acute rheumatism, anæmia is a symptom which is virtually constant.

These two maladies, then, are in every way widely different from each other. It remains to be considered whether the other two are equally distinct or whether any relationship can be traced either to one another or to those which we have already discussed. It may here be noticed that Weichselbaum, who has devoted much care to the study of the pathological conditions found in the senile form [7], has come to the conclusion that they differ only in degree from those of the classical

type of arthritis deformans. Clinically, too, one meets every now and then with patients of middle age suffering from a joint affection which neither by its localisation nor its course is to be distinguished from the senile malady, which it moreover resembles in that pain and other general symptoms are notably absent.

Arthritis deformans, acute and chronic.—Having dealt with these affections of the extremes of life, we have to consider the two remaining joint diseases which I have mentioned. It is here that the differential difficulties become most marked. In the paper previously referred to Dr. Hale White makes out a strong case against the identity of the two maladies, but yet I must confess that on carefully reviewing the evidence, I am by no means convinced that we are able as yet certainly to divorce them. That both of them are distinct from true rheumatism may, I think, be taken as definitely established, though some cases which have been described as acute rheumatoid arthritis appear actually to have been rheumatic or due to some other recognisable infection. The most striking difference between the two forms appears at first to be in the rapidity of their course. The acute disease often cripples its victim in a few weeks or months, while the course of the chronic may be reckoned in years or even decades. But the former does not always act with such terrible suddenness, crystallising the patient, so to speak, in a single attack. It may be jerkily progressive, coming on in successive bouts, each accompanied by pyrexia and followed by increased maiming. So also the course of chronic arthritis deformans may ebb and flow, and a slight degree of fever is not infrequent in the exacerbations. In my experience this is more common in younger women—those, for instance, in whom the disease begins at about the age of 30. It seems to me, therefore, that in this respect no sharp gulf unbridged by intermediate cases separates the acute and chronic forms. As regards the incidence upon the joints, there is a distinct and fairly constant difference. In the acute variety many joints are affected at much about the same time, and amongst these the proximal interphalangeal and temporo-maxillary are usually conspicuous. The brunt of the chronic affection falls upon the metacarpo-phalangeal and it spreads in a slow march to others, the knees being usually involved early. The character of the deformity hence differs in the two cases; this difference is accentuated by that in the anatomical changes which will be referred to later. I have here photographs and skiagrams of a typical case occur-

ring thirteen years ago in a girl who was then 10 years old.* Within a few months every joint in the body was probably more or less damaged; the attack was a single one and the condition has since remained, to all intents and purposes, stationary. It seems to me possible that the difference in distribution between the acute and chronic conditions may be one of degree only; in the former the process is too rapid for any marked selection to occur and the lesions are accordingly more generalised. But this is only a suggestion; the fact that there is a difference in distribution remains. Some stress has been laid upon the fact that in the acute affection there are commonly fusiform swellings upon the interphalangeal joints; to this I attach no importance, as this shape is that naturally assumed in any acute or subacute inflammation of these joints and is frequently seen in gout, in acute rheumatism, and in the exacerbations of chronic arthritis deformans.

A much more striking point of difference is met with when we come to consider the question of morbid anatomy. The inflammatory changes in acute arthritis deformans are primarily and mainly in the synovial membrane. The cartilages are damaged secondarily, and the only change which can be demonstrated in the bones is the marked thinning of their ends to which Dr. Hale White has called attention and which is so very conspicuous in the skiagrams which I am showing to-night. In the chronic form, on the other hand, degenerative changes in the cartilage, involving fibrillation, patches of softening, and hyperplasia are the chief features in the pathological picture. At the same time, or soon after, the subjacent bone is absorbed in some places and hypertrophied in others. The synovial membrane, however, does not escape; it is usually hypertrophied and may be sufficiently inflamed to cause marked dropsy of the joint. It may, indeed, be suggested that the difference between the two types is not so great as at first appears. As age increases the patient becomes progressively less liable in general to chronic synovial disease other than traumatic. Thus, for instance, the incidence of tuberculous disease of the knee is in childhood mainly upon the synovial membrane, in adult life upon the bone-ends. It therefore occurs to me that the contrast in arthritis deformans may be only a seeming one, and that the acute and chronic types may be the expressions merely of a similar process attacking joints at different ages. It is quite exceptional for what I may call

* For permission to refer to this case I am indebted to the kind courtesy of Dr. Allchin.

the adult type of the disease to be seen in childhood; and both types show the common and perhaps essential features of absorption of bone and overgrowth of synovial membrane.

One other point still requires explanation. There can be no doubt that in many, if not most, cases of chronic arthritis deformans the nervous system is profoundly affected. I am not quite able to follow those who regard the affection as a nervous disease; the view that it is a cerebro-spinal toxæmia has been ably argued by Dr. Llewellyn Jones [8], but without carrying conviction to my mind. Nevertheless we often find muscular, cutaneous, and vascular trophoneuroses, exaggerated reflexes, and even neuritis in association with chronic arthritis deformans. Mental worry seems to be a predisposing factor in some instances and certainly often determines exacerbations; sometimes, too, in youngish women, the onset of the disease is associated with severe depression and a premature menopause. But we often see it in a most intense form without a trace of neurosis, and it hence seems to me more likely that the latter is more probably a concomitant effect of the specific toxin or infective virus. In acute arthritis deformans the nervous system is but little affected. It is true that in most of the long-standing cases which I have seen the patient has been "neurotic," but not more so than might have been anticipated from her crippled and invalid condition; objective signs of nervous disease are as a rule absent. On the whole I am not inclined to regard this dissimilarity as connoting an essential difference between the two conditions. If, as I think probable, they are due to infective or toxæmic processes of similar nature, it is only reasonable to expect that the more acute would be characterised by pyrexia, while the prolonged action of the virus would involve more especially the nervous system.

In other words, if we exclude doubtful cases, in which known infective causes may be present, the points of similarity between acute and chronic arthritis deformans seem to me to outweigh their differences and to be compatible with the possibility that they represent the acute or inflammatory and chronic or degenerative phases of the same malady. Intermediate forms occur within the experience of all those who have the opportunity of seeing a considerable series of cases and go to negative the drawing of a hard and fast line of separation.

The senile form is, to my mind, essentially degenerative, and may therefore, in my opinion, be the result of various

factors, acting alone or in combination. Among these is, no doubt, to be reckoned the ordinary type of arthritis deformans, which may pass on into it, just as the large white kidney may —somewhat rarely, perhaps—undergo contraction and fibrosis. In many instances there is no precedent arthritis deformans, and in such cases we have to look to other etiological agencies, in particular, old age, as the causes of degeneration [9]; it must be remarked, too, that in cases in which permanent joint affections have been produced by true rheumatism, the advent of old age is liable to bring on senile changes indistinguishable from typical osteo-arthritis.

Finally, the disease described by Dr. Still is an infective process which appears to be in no way related to any of the other forms.

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XX.—*A Method of ascertaining the Position and Size of the Stomach by means of the X-Rays and a Tube filled with Subnitrate of Bismuth.* By Norman Dalton, M.D., F.R.C.P., and A. D. Reid, M.R.C.S., L.R.C.P. *Read March* 24, 1905.

My attention was first drawn to this subject by reading that if the stomach were filled with an emulsion containing subnitrate of bismuth in suspension, its outline could be detected by placing a Crookes' tube behind the patient and examining the abdomen through the fluorescent screen. It occurred to me that the amount of emulsion required to fill a dilated stomach would be inconveniently large, so that if it really filled the stomach it must, by its weight, displace or distort that viscus, while, if it did not fill it, no idea of the shape or size would be obtained. I next inspected tubes filled with metals (lead or iron) which the instrument-makers were supplying. These appeared to me to be insufficiently flexible, and therefore dangerous. It then occurred to me that an ordinary stomach-tube filled with powdered subnitrate of bismuth would show well when passed into the stomach and illuminated by the X-rays, and would probably adapt itself to the outline of the greater curvature. I found by experiment outside the body that such a tube was as opaque to the rays as a tube containing mercury. I then passed the tube into the stomach and found that it could be distinctly seen through the screen when the X-rays were turned on behind the patient.

This is the tube originally used. It is as flexible as an empty stomach-tube, but rather heavier. It is a little thicker than necessary, but that is no drawback to its use, as a thick tube irritates the œsophagus less than a thin one, and, as we afterwards found, it glides along the stomach more readily than one of smaller calibre. The one drawback is that it is too short. The reason for this is that neither instrument-makers nor the manufacturers of rubber goods possess a mould which is as long as I could wish. We have had a piece joined on to it, but the roughness at the joint prevents the added part from being passed beyond the soft palate. Still, the additional part is useful as a handle, and we gain an inch or two by it. It is rather a difficult matter to fill the tube with the powder evenly, so that there are no crevices, but

with care, and by frequently examining it with the rays and screen during the filling process, it can be done.

In the vast majority of cases patients have not found the use of the bismuth tube more inconvenient than that of the ordinary tube. When they have complained the inconvenience appears to have been chiefly due to the fact that the tube must be passed while the patient is in the recumbent posture. I may say here that I have only used the tube in those cases in which an ordinary tube would be used, either for diagnosis or treatment. When there was reason to suspect cancer of the body of the stomach or a recent ulcer I have left it alone. In fact, the absence of tenderness over the stomach area and the great desirability of obtaining a definite diagnosis (mostly with a view to operation) have been the chief points which have decided me to use it. I have heard of accidents occurring with the empty tube, and I would plead extreme caution as the reason why I have so few cases to show to-night.

I first used the tube in 1901. We could see its movements and the final position which it took up very clearly through the screen, but we could not reproduce it in a way which would enable us to demonstrate these points until Mr. Reid succeeded in taking photographs of it *in situ*. The method used at present is as follows: The ordinary stomach-tube is passed once a day for two or three days beforehand in order to habituate the patient to its presence in the stomach. On this point I would remark that patients whose stomachs have become dilated by pyloric stricture or by displacement tolerate the tube well, and they are usually thin, which enables the tube to be well seen through the abdominal wall. If the examination is to be made at two o'clock, they are given no food after nine, or the stomach can be washed out at about one o'clock. A metal disc is then attached to the umbilicus by plaster and a metal wire fixed along the curve of the lower ribs by the same means. The patient then lies on his back on the couch, beneath which is the Crookes' tube, the arrangement of which will be explained by Mr. Reid. The tube is then passed by an assistant, while the epigastrium is examined through the fluorescent screen. As the tube reaches the stomach it can be seen to emerge from beneath the ribs, rather to the left of the ensiform cartilage. It then passes straight downwards until it reaches the greater curvature. We have learnt, as I will show, that on reaching this point it carries the wall of the stomach a little way downwards. Then, however, the tube becomes convex to the left, while the point

runs upwards and to the right, describing a curve which corresponds to the pyloric end of the greater curvature. As the tube ceases to be vertical the depression of the greater curvature disappears. The point is finally arrested at or near the pylorus, and then the tube bulges out more to the left and apparently lies on the lower and left parts of the greater curvature. It now gives what I believe to be an accurate outline of the greater curvature from the pylorus to near the cardia. When the tube is being withdrawn the curve which corresponds to the fundus first becomes straightened, then the point is observed to slide down the pyloric end, and if the stomach be dilated at a certain point it swings free, the part of the tube still in the stomach becomes straight, and finally disappears beneath the ribs.

I have endeavoured to describe what is actually seen. It would have been more convincing to have demonstrated it, but I think that you will agree with me that it would not have been justifiable to bring a patient here. In the first place, the examination should only be made for purposes of diagnosis, and secondly it would be impossible for more than a few persons to see the tube at a time, and the prolongation of such a process is, of course, undesirable. Fortunately, Mr. Reid's photographs show the main point, viz. the tube *in situ*, and I will now show you the lantern slides taken from these photographs. I have the original photographs with me here for comparison with the slides. Some of the cases which I will show have been operated upon, and at the operation I was able to see that the position of the tube in the photograph really corresponded to the greater curvature of the stomach.

I have not been able to obtain a specimen of a normal stomach. I did not like to experiment on healthy people, and experiments on the cadaver failed in a way which I shall mention later. It is obvious, too, that, as so much of a normal stomach lies behind the ribs, left lobe of the liver, etc., it would not be easy to see the tube behind these structures unless the patient were extremely thin. I recently examined a very obscure case with the bismuth tube and X-rays, and we were able to see the tube dimly through the screen, and its position indicated that the stomach was normal in size, shape, and position. But the photograph was not successful, so I cannot show the appearance which the tube presented. This patient was afterwards operated on, and the stomach was found to be in every way normal, but there was a large malignant tumour growing from the vertebræ behind it. It would,

I believe, be almost impossible to see the tube in a contracted stomach, especially as contracted stomachs are often very irritable.

The first four slides which I shall show are from the same case, and they show the tube in different positions. No. 1 is, unfortunately, rather indistinct, because the tube in that position has so much tissue in front of it. However, in the photograph it shows the tube as it first makes its appearance rather to the left of the middle line. The white disc marks the umbilicus, and the white lines follow the curve of the lower ribs in all of the slides.

No. 2 shows the tube as it is arrested for a second at the greater curvature. Its point is as low as the umbilicus.

No. 3 shows the tube bent to the left and with its point ascending to the pylorus. It will be seen that the pyloric end is pushed to the right by the tube as it advances. It must be noted that the middle of the tube is now above the umbilicus, whereas in the last slide the point was at the umbilicus. The difference shows the extent to which the greater curvature was depressed by the tube while straight, and this gives, I believe, an estimate of the tonicity of the stomach wall.

No. 4, as you will see by the difference in the wire which marks the ribs, was taken on a different day from the other three, but it is from the same case. It shows the point of the tube, no longer pushing the pyloric end to the right, but arrested at the pylorus itself. The rest of the tube has now applied itself to the greater curvature, the pyloric end of which is lower than and well to the right of the umbilicus. The fundal end is not bulged out to the left, showing that there was no great dilatation. The diagnosis which I made was gastroptosis. This patient elected to have a gastro-jejunostomy performed, and at the operation the pyloric end of the stomach was found to be exactly as delineated by the tube as seen in the slide. There was no ulcer, adhesion, or cancer.

No. 5 is from a woman whom I have known as a patient for over twenty years. She is now about 43. All this time she has suffered from dyspepsia, associated with horrible epigastric and præcordial sensations, and with remarkable manifestations of hysteria. Her right kidney is mobile. She is, in fact, a typical case of general enteroptosis, or Glénard's disease. It will be seen that the position of the tube indicates a very marked degree of gastroptosis, the pylorus being displaced downwards and to the right, and the pyloric end of the greater curvature being well below the umbilicus. It

differs from the last case in the fact that the tube bulges far to the left, showing that there is gastrectasis as well as gastroptosis, which is the usual state of affairs in old-standing cases of Glénard's disease. The position of the stomach has not in this case been verified by operation, but I do not think that there can be much doubt about the diagnosis.

No. 6 shows a dilated stomach. It will be seen here that the curve is quite different from that in the cases of gastroptosis. It is a larger or more open curve, the pyloric end does not descend so low, and the fundus is well bulged out towards the left. At the operation the greater curvature was found to correspond with the outline shown on the slide. The cause of the dilatation proved to be a number of adhesions around the pylorus, probably the result of previous ulceration.

No. 7 shows, I believe, a very dilated stomach. The low position of the greater curvature is general and not localised to the pyloric end as in cases of gastroptosis, and the left side is well bulged out. This case has not yet been operated upon.

No. 8 is of great interest. The tube became arrested behind the umbilicus in the position seen in the slide and it would go no further. When the patient was operated upon, it was found that the pylorus was actually near the umbilicus, rather to the right of the point of the tube. It was bound down there by a mass of adhesions. I was inclined to conclude from this that the point of the tube would always reach the pylorus, and would always enable us to tell where the pylorus was. This conclusion proved to be incorrect; for shortly afterwards the tube was passed into the stomach of a girl who was believed to be suffering from hysterical vomiting, and it was arrested, as in the case just mentioned, near the umbilicus, and would go no further. A few days later we examined this girl with a somewhat thicker tube. This time it was again arrested at the umbilicus for a few seconds, but then the point ran up to the pylorus in the usual manner, and the curve finally obtained showed a moderate dilatation of the stomach. I then passed the tube into the stomach of a dead man, and found through the screen that it was arrested at the umbilicus, just as in the last two cases which I have mentioned, and it could not be got to move further. On opening the abdomen it was seen that the reason why the tube would not go on was that its end carried the stomach wall before it, the wall covering the tube like the finger of a glove. The great difference (from the present point of view) between a living and a dead stomach is that the latter is quite atonic and gives before

the slightest pressure. It would, therefore, appear that if, in any case, the tube becomes permanently arrested at about the umbilicus it may mean that the pylorus is really there (as in the first case) or that the stomach is very atonic. I have already said that I think the difference between the lowest point reached by the end of the tube when it is straight on the one hand, and the transverse line marked by the tube in its final position on the other hand, gives an estimate of the tonicity of the stomach. I may say that if the point of the tube becomes arrested, retching on the part of the patient or even coughing may disengage it and permit of its gliding up to the pylorus.

No. 9 is merely to show the great flexibility of the tube. It is from the case of the girl, supposed to have hysterical vomiting, in whom the tube was arrested at the umbilicus. The middle of the tube became coiled, as is seen, within the stomach, without causing the patient any discomfort.

No. 10 is from a case of general enteroptosis. In its main characteristics it resembles the other two cases of gastroptosis.

I have not been able to obtain a case of hour-glass contraction, but I fear that the tube would not ascend the right side of the proximal compartment. If it did we might get an interesting curve, probably with the point of the tube close to the upper part of the stem.

In conclusion, I may say that I think that the method is safe, provided that we only use it in those cases in which we think the use of an empty stomach-tube justifiable. I also think that it gives a very accurate estimate of the size and position of the stomach, and probably of the shape of the viscus as well. I thought it possible that the outline obtained would be greater than the real outline of the stomach because of the divergence of the rays, but Mr. Reid assured me that the difference could only be slight, and I verified this at several operations besides those I have mentioned to-night. It is also possible that the point of the tube does not always reach the pylorus, a result of which we know would be that the pylorus would appear further to the right than it really is. Even allowing that the size appear rather greater than the actual one, and that the pylorus be not always reached, we nevertheless get a valuable indication of the physical condition of the stomach. The method certainly enables one to recommend an operation with great confidence. It has this advantage over the introduction of air or water into the stomach, viz. that it does not stretch the stomach in any way, and I believe that there are stomachs which are

more dilatable than dilated. Water, too, by its weight may displace or distort the stomach, and both air and water may escape through the pylorus. None of these objections apply to the bismuth tube, which gives the outline of a perfectly empty stomach; and finally, it has this great advantage, that if the patient should complain of pain it can be removed from the stomach more rapidly than either air or water.

The technique of taking the skiagrams was as follows: The patient is laid on his back on a canvas-topped couch, underneath which the tube is mounted on a double slide, which allows movement longitudinally and transversely, thus rendering it easy to centre it under any given spot. The tube is enclosed in an opaque box with an adjustable circular diaphragm placed over a 3-inch aperture opposite the anode, the smallest diaphragm being used that will include the required area. When the bismuth tube has been inserted as far as it will go, the patient is directed to hold his breath, and the exposure of from 10 to 20 seconds made, the plate being placed on the abdomen. The apparatus must be powerful to enable a sufficiently good skiagram to be taken in this time; that used in taking those shown consisted of a 10-inch variable primary coil worked off the 100-volt continuous main, with an electrolytic interrupter working at 15 ampères on the primary of the coil, and either a heavy anode or water-cooled tube.

DESCRIPTION OF PLATE IV.

Illustrating Dr. Norman Dalton's paper on "A Method of ascertaining the Position and Size of the Stomach by means of the X-rays and a Tube filled with Subnitrate of Bismuth."

N.B.—All the illustrations are negatives, and in all the white disc marks the umbilicus, and the white lines the curve of the ribs. The tube is also white.

A.—Gastroptosis without gastrectasis at the fundus. This is described in the paper as No. 4 of the series of lantern slides.

B.—Extreme gastroptosis with gastrectasis at fundus. No. 5 in the paper.

FIG. A.

FIG. B.

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DESCRIPTION OF PLATE V.

Illustrating Dr. Norman Dalton's paper on "A Method of ascertaining the Position and Size of the Stomach by means of the X-rays and a Tube filled with Subnitrate of Bismuth."

C.—Gastrectasis from adhesions at the pylorus. No. 6 in the paper.

D.—Extreme gastrectasis. No. 7 in the paper.

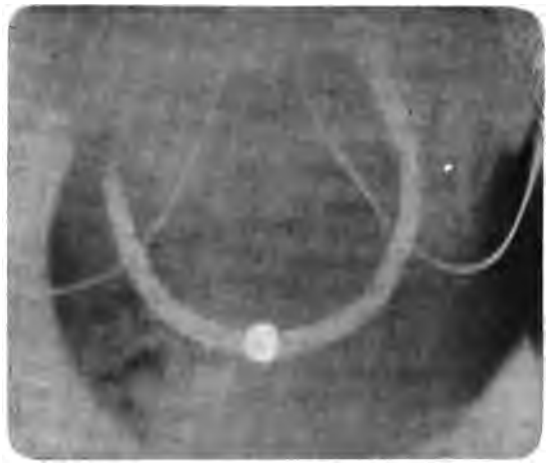

FIG. C.

FIG. D.

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XXI.—*Three Cases of Stenosis of the Ureter.* By Douglas Drew, B.S., F.R.C.S. *Read March* 24, 1905.

CASE 1. *Hydronephrosis; pyelo-ureteroplasty; cure.*—E. E., æt. 34. Mother of four children; was admitted under my care on May 13, 1903, complaining of pain and swelling in the left side of the abdomen. The pain was of a dragging character, but was not increased by walking. The swelling had been present four years and had gradually increased in size. There was no history of hæmaturia or other urinary symptoms except slight frequency. The left loin was occupied by a well-defined elastic and smooth swelling, having all the characters of a renal tumour. It moved with respiration, and was not tender on manipulation. The urine was normal.

Operation: May 23, 1903.—The kidney was exposed through an oblique incision in the loin, and, after emptying it of about three quarters of a pint of urinous fluid, it was dislocated on to the loin, and the cortex was freely incised. The calyces were carefully explored by the finger, but no stone or other abnormality was found, and as the pelvis was too much dilated to permit of its being explored through the incision in the convex border of the kidney, the pelvis was incised vertically for two inches on its posterior aspect. The ureter was isolated, and was found to be very much narrowed for a distance of about a quarter of an inch at its junction with the pelvis.

By holding the edges of the incision in the pelvis open I succeeded, after some difficulty, in passing a probe into the ureter, the orifice of which was very small. It was situated about three quarters of an inch above the lowest part of the distended pelvis, and it seemed probable that the distended portion of the pelvis had exercised some pressure on the ureter and rendered the obstruction of it complete.

The narrowing of the ureter was not of a cicatricial nature, but was evidently congenital, and the cause of the hydronephrosis.

The narrow part of the ureter and adjacent part of the pelvis were divided vertically downwards for a distance of two thirds of an inch by inserting the blade of a pointed pair of scissors into the ureteral orifice. The adjacent edges of the pelvis and ureter were then accurately united with the finest catgut in a round needle, the stitches being inserted

from the interior of the pelvis. After this had been completed, a No. 7 English scale catheter was passed from the kidney down the ureter into the bladder, and then withdrawn, leaving about two inches in the ureter.

The incision in the pelvis was next sutured with fine catgut, and the incision in the cortex was closed around the catheter with stout catgut and the organ replaced. A drainage-tube was inserted into the kidney and the abdominal wound closed. The catheter was withdrawn at the end of thirty-six hours, after which a little blood was found in the urine for some days. The wound healed by first intention.

On the eleventh day a slight phlebitis, with œdema, developed in the left leg, and this somewhat delayed convalescence.

Patient was discharged well on June 27.

I have seen the patient at frequent intervals up to May, 1904, and she has had no further trouble, and the left kidney cannot be felt.

Case 2. *Hydronephrosis, congenital narrowing of the upper part (4 inches) of ureter, plastic operation.*—M. K., æt. 50, mother of nine children, was admitted under my care on January 27, 1905, complaining of pain and a swelling in the left side of the abdomen.

The tumour was noticed accidentally a year and nine months before, and at first did not cause any trouble; more recently there had been much pain of an aching character. The tumour had steadily increased in size. The only other symptom was some frequency of micturition.

Operation, February 2.—The kidney was exposed by the oblique incision, and emptied of 1¼ pints of urinous fluid by a trochar, after which the organ was dislocated on to the loin, and the opening into the pelvis was enlarged, and the interior explored by the finger. The kidney was very little affected by the distension.

The upper four inches of ureter were isolated and found to be very much narrower than normal. The incision in the pelvis was enlarged, and the ureteral orifice sought for; it was situated at the lowest part of the pelvis and just admitted a large size metal probe. It was obviously impossible to remedy the condition of the ureter; at the same time, the condition of the kidney was such that it did not appear desirable to remove it, and it was thought that by enlarging the ureteral orifice it might possibly cure the hydronephrosis. This was

accordingly done in the same manner as in Case 1, and an elliptical portion of the dilated pelvis was excised and sutured. The wound healed satisfactorily, but three weeks after the operation the tumour had again appeared, and will probably require nephrectomy as it is causing pain.

Since writing the above, I have seen the patient. The swelling has now (May 8th, 1905) almost disappeared, although tenderness in the loin persists.

Case 3.—*Pyonephrosis, due to stones in the ureter; nephrotomy and removal of stones; subsequent nephrectomy.*—A. P., æt. 28, recently married, was admitted to the hospital under my care August 2, 1904, looking emaciated and ill. The temperature was 90°.

The right side of the abdomen was occupied by a large elastic tumour, extending forwards beyond the middle line, and having all the characters of a renal tumour. The urine was acid, specific gravity 1025, and contained no albumen or pus. The tumour caused continuous aching pain in the side, which was aggravated by walking about.

The onset of the trouble appears to have dated back about ten weeks, when she had an attack of pain in the right side and other symptoms, which were at first thought to be indicative of influenza.

The swelling in the abdomen appeared shortly afterwards, and was associated with some pain and frequency of micturition, and the passage of "thick" urine.

August 5, *operation.*—The kidney was exposed through the lumbar incision. On incising it a large quantity of thin pus escaped. It was partially isolated and drawn up on to the loin, and the interior thoroughly examined, but no stone was found, or other cause to explain the condition. The pelvis was traced downwards to the ureter, and on isolating the latter a fusiform swelling was found on it about an inch from the pelvis. On incising the swelling longitudinally, two small collections of pus were opened in the wall of the ureter, and the interior was lined by granulation tissue, embedded in which were three minute calculi, which were removed. A large drainage-tube was inserted into the kidney, and a second one was passed down to the opening in the ureter, and the abdominal wound closed around.

The patient suffered severely from shock after the operation, and on the following day, as shock persisted, intra-venous injection of saline was resorted to, with decided improvement.

The drainage of the kidney was continued for three months. At the end of two months, as the quantity of discharge had gradually decreased, the tube was omitted, but it had to be replaced in a few days, owing to a rise of temperature, and upon so replacing the tube a quantity of pus escaped.

Throughout the whole period the urine did not contain any pus, and only on one occasion was albumen present as a faint cloud. It was therefore evident that the ureter was completely blocked by the cicatrisation of the ulcerated surface; so, at the end of three months (November 4) I decided to explore the ureter in the hope that I might be able to remedy the stenosis and possibly save the kidney, but it soon became evident from the condition of the kidney that any plastic operation was out of the question, so nephrectomy was performed.

The nephrectomy proved unusually difficult, owing to the height the kidney extended above the twelfth rib, and the extreme density of the adhesions. In separating the upper part a hole was made in the peritoneum, which was at once closed; the kidney and upper part of the ureter were removed.

The after-history was uneventful; the healing was slow, owing to the septic condition at the time of operation.

Specimen.—The specimen shows the dilated kidney and the narrowing of the upper end of the ureter. Before the latter was split open a fine probe would not pass through the narrow part. The calculi are composed of (?) oxalates.

These three cases are examples of the rarer conditions that may give rise to hydro- or pyonephrosis, and illustrate the importance of examining the ureter where the cause of the distension is not evident. Cases of hydronephrosis caused by some congenital narrowing of the ureter are very uncommon.

Until recent years it was not customary to explore the ureter with the object of discovering and remedying any such abnormality. Cases of hydronephrosis were either treated by primary nephrectomy or by nephrotomy and drainage, and the underlying cause remained undetermined. Occasionally a case thus treated resulted in a cure. When this happened, it was more than probable that the hydronephrosis arose from kinking of the ureter, but the majority of the cases after nephrotomy were left with a urinary fistula, which necessitated a subsequent nephrectomy for its cure.

In the first case under notice, although the renal tissue was considerably damaged, yet it was obviously a good organ,

and it would not have been justifiable to have removed it. In the second case the kidney appeared to be almost normal in size, although the pelvis contained 1¼ parts of fluid.

With regard to the method of suturing adopted, the walls of the ureter and kidney were so thin that it was quite impossible to stitch them together without passing the sutures through the entire thickness of the walls, and as this could be done more easily from the interior of the pelvis, this course was adopted.

The chief point of interest in the third case is that I was fortunate enough to find, and to be able to observe, the condition which subsequently produced the stenosis. At the time I expressed the opinion that stenosis would almost certainly result, but apart from the remote possibility of the patient escaping without further trouble, her condition rendered a primary nephrectomy out of the question.

From a pathological point of view the case is of interest, in that it is difficult to offer any satisfactory explanation of the presence of the small calculi at this particular spot in the ureter, unless some narrowing of the ureter existed. It appears to me to be improbable that the calculi descended from the kidney, as if this had occurred they would not be likely to lodge in the ureter, being so small, and other calculi would in all probability have been found in the kidney.

Occasionally it happens that no cause can be discovered in the ureter or elsewhere for the occurrence of a pyonephrosis. Within the last few months I have had an example of this kind in a young woman. The tumour was noticed by her doctor during the earlier months of pregnancy, and as the abdomen enlarged it apparently disappeared. She came under my care about six weeks after her confinement, which was normal, with a very large pyonephrosis. Nephrotomy was performed, and after draining for about two and a half months the wound healed, and she is now quite well. I mention this case as I am at a loss to offer a satisfactory explanation of it.

XXII.—*Three Cases illustrating the Condition of the Small Intestine some years after extensive Enterectomies.* By A. E. Barker. *Read March* 24, 1905.

THERE must be few surgeons now who do not recognise the peculiarly disastrous effects of paralysis of the bowel in advanced intestinal obstruction. These make themselves evident to the operator in many ways before, during, and after the relief of the obstruction, and are, unfortunately, so often seen that they require no comment here. But few of us are likely to have many opportunities of seeing the effects of this paresis on the *living* gut *years* after the relief of severe obstruction. Three such opportunities have fallen to my lot quite recently, and it seems desirable that they should be put on record, emphasising, as they do, some points of fundamental importance in the treatment of acute intestinal obstruction.

The two first patients concerned were both women. They were admitted some years ago for gangrenous femoral herniæ and in both I was obliged to resect long tracts of small intestine in order to obtain relatively sound tissue for the anastomosis. Both made a satisfactory recovery from these operations but were readmitted in October, 1904, for operations in the course of which it was possible to examine the original seat of the enterectomies. In both it was most interesting and instructive to observe that, though the communication between the two portions of the bowel was perfect and as wide as possible, the afferent or proximal portion still showed unmistakable signs that it had not yet recovered its former tone as contrasted with the efferent or distal portion, which was normal. The significance of this observation appears to me very great both from a medical and a surgical point of view, and so far as I know has not yet attracted attention at home or abroad.

A description of the first operation in each case has already been published (*vide infra*), and it is only necessary here to state that, in the first, 18 inches of small gut were removed in June, 1899, on account of a gangrenous femoral hernia. This was done through the femoral ring, Poupart's ligament being divided to permit of the reduction of the anastomosed bowel and ligatured mesentery into the abdomen. The junction was "end-to-end." In the second case, also of gangrenous femoral

hernia but in a much graver condition, the gangrenous loop was on February 23, 1903, cut away below the femoral ring, and then the ligatured ends were passed into the abdomen and brought out through a median incision, where six and a half feet of damaged small intestine were taken away from the proximal portion and a *lateral* anastomosis was made.

Case 1 was *re*admitted on October 24, 1904, for the radical cure of double hernia—*i. e.* five and a half years after the enterectomy. A large femoral hernia was found to exist on the left side in the seat of the first operation, and a moderate-sized inguinal hernia on the right. The patient was very stout. The left hernia contained much intestine, the movements of which could be seen through the thin coverings, and was obviously due to the weakening of Poupart's ligament by its division in the earlier operation. On October 27 the usual operation was done for the radical cure of this hernia.

When the sac was opened I took the opportunity of examining the contained bowel before it was reduced, in the hope of finding the anastomosis. Fortunately, it was there, and its condition could be demonstrated to a large number of those present in the operating theatre. When drawn forward and laid on a layer of white gauze, we could see that the two portions lay accurately in line, and that the junction was as smooth as the rest of the bowel, and without a trace of adhesion to any other structure. It was marked by faint white streaks indicating the seat of the sutures, all of which had completely disappeared. There was not the slightest sign of contraction or narrowing. But what struck us all most forcibly was that all the visible bowel distal to the anastomosis was in a state of very active vermicular movement and was contracted to a small size, while that exposed above the junction was smooth, sluggish, somewhat thinner, and of considerably larger lumen than that below. I drew the coil forward and stretched it gently, but this made no difference in the relative sizes of the two portions. It was, in short, evident that the original paralysis of the bowel above the obstruction had left behind it a certain want of tone in the muscular wall of the proximal portion, or, blunted nervous irritability. The only other trace of the original operation was a tiny adhesion of an adjacent coil to the mesentery close by, which I did not think it worth while to divide. After clearing up these points of interest, the bowel was reduced, and the radical cure was completed. The other inguinal hernia was operated on a

week later, on which occasion the first was found blamelessly healed *per primam*. The patient left the hospital well on November 26.

Case 2 was *re*admitted on October 14, 1904, suffering from intestinal obstruction of some days' standing. Suspecting a band of adhesion from the former operation in February, 1903, I opened the abdomen above the median scar and found several very firm adhesions between the stump of the mesentery and the lower end of the scar. Round these adhesions some coils were kinked. These were released, and I drew out the intestine and obtained a good view of the old lateral anastomosis of twenty-one months before. Here, too, it was extremely interesting to note that though the opening between the two portions was widely patent, the proximal, as far up as it was exposed, was of considerably larger size than the distal. Both were contractile, but the lower more so than the upper. Several adhesions were left undisturbed, and the abdomen was closed. Convalescence was uneventful, and the patient left hospital quite well on November 7.

These two observations made *intra vitam* on patients suffering from no general disease and quite healthy at the time except for the local condition, fill up to a certain extent a gap in our knowledge as to the changes which take place in an intestine above an acute obstruction, and as to the period during which these effects may remain after the latter has been relieved. It might at first sight appear almost incredible that evidence of want of tone in the bowel should exist several years after the cause has been removed. But we see the same thing elsewhere frequently. For instance, who does not know the cold congested appearance of a limb which perhaps months or even years before has been the seat of some inflammatory septic condition such as erysipelas? In this case the tissues have been saturated with toxins just as the walls of the intestines have been, and the result has been atony of the coats of the vessels of the part and deficient local nerve energy, or even paræsthesia.

In Case 1 it was most striking to see the distal portion of the gut wriggling, and contracting energetically under the stimulus of exposure to the air, while the proximal part down to the line of anastomosis was sluggish and almost quite indifferent to the same stimulus. And when we come to reflect upon what actually takes place in the walls of the intestine above an acute strangulation, we must admit that there is little cause for wonder if the effects should last for a long time.

Should the original obstruction remain unrelieved for long, we all know the dilatation of the gut above which follows, accompanied at first by increased peristalsis, but terminating before long in paresis or complete paralysis. This paralysis is probably not entirely due to exhaustion of the muscular coats from overstrain, but is the result of the saturation of all the tunics of the bowel with toxins developed in the fermenting contents. That this is so seems to me proven by the fact which every surgeon must have observed over and over again—*i. e.* that when a bowel is examined above a bad obstruction it will be found in many cases œdematous for several feet on the proximal side of the strangulation. This need not be due to the presence of ulceration at the point. This ulceration, which is so common in the bowel just above an obstruction and for a foot or two upwards, might explain the œdema locally. But far above this the œdema extends where no breach of surface can be detected, at all events with the naked eye.

In Case 2 I found œdema more than six feet above the strangulation when I was making the original anastomosis at that point. We see, also, further evidence in a submucous ecchymosis associated with the œdema. In another case, where I removed five and a half feet of small intestine for acute strangulation some years ago, the œdema was so marked in the proximal portion while the "end-to-end" anastomosis was being made, that I was in serious doubt as to whether the vitality of the bowel was sufficient for repair. Happily it proved to be so, and the patient made an excellent and uninterrupted recovery. That such toxins are intensely irritating on other surfaces is well seen on the skin soon after an artificial anus has been made to relieve a bowel damaged by acute obstruction. In a case recently operated on for this condition the ulceration of nearly the whole of the abdominal surface was the most formidable complication in the subsequent treatment of the case. Again, the œdema is *not* seen in the earlier stages of acute obstruction before the ferment toxins are developed in the retained contents, but it is found in neglected cases in increased intensity and extent in proportion to the duration of the obstruction, and possibly also in relation to the quality of the aliment previously given to the patient. But though I have been for years alive to these changes in the bowel wall and have planned my resections and "short-circuit" operations with due regard to them, I confess I was hardly prepared to find evidence of more or less paresis of the proximal part *some years* after the obstruction had been relieved.

This has all to be taken into account also in cases where we elect to perform a short circuit round an obstruction, as Case 3 shows, which has come under notice since this paper was sent in to the Society. This was a case of a man, æt. 29, who was admitted into hospital some two years ago for acute obstruction grafted on a chronic condition which we diagnosed to be probably cancerous in the hepatic flexure of the colon. He had stercoraceous vomiting and much distension, etc. I opened the abdomen below the navel, and found greatly distended small bowel. This was slit open over a basin and gave exit to large quantities of gas and fluid fæces. It was then possible to explore further, and I found the cæcum and ascending colon distended, and the descending colon and sigmoid flexure empty and shrunken. The obstruction, therefore, was obviously between the two regions. I then united the lower part of the ileum to the sigmoid flexure side to side, and closed the evacuation opening, as he was too ill for further operation. Recovery followed, but the man would not stop in hospital for the resection of the growth, but promised to return as soon as he had arranged his business affairs. However, he did not appear until about three weeks ago. When I opened the abdomen last Thursday week the growth was found too extensive and fixed under the liver to justify removal, and I closed the wound. But before doing so the "side-to-side" junction was examined, and the place where the evacuation opening had been made and closed. Both were in perfect condition, no traces of the black thread sutures being seen, and not the smallest suspicion of adhesions to surrounding parts where the bowel had been stitched and manipulated; all was smooth over the white-lined cicatrices. Nevertheless, as in the other cases, the proximal, and in this case the distal, portion between the anastomosis and the cæcum, about eight or ten inches, were still in a state of atony, and contrasted markedly with the upper part of the ileum and jejunum, which could be seen contracting in active vermicular action. The man's health had been excellent since the first operation, and his bowels quite regular and motions normal. Incidentally I may express the regret that this poor fellow did not redeem his promise of returning for the removal of the growth before it grew too extensive and fixed for excision. For he is still in good condition, though two years have elapsed, but is now losing colour and strength from generalisation, and complains of nothing else.

When we find such changes in the intestines as those just described even *years* after the case has been relieved, we are

more than ever driven to the conclusion that in acute strangulation we must operate before they can have had time to develop. And if this be impossible we must not hesitate where resection is necessary to remove large tracts of bowel in order to get well above the paralysed portion, saturated as it is with toxins and thus devitalised.

In another respect the first two cases offer us a valuable hint by contrast. In Case 1 the resection was performed through the femoral ring, and the difficulty of doing so was well exemplified. It was not so much the "end-to-end" suture which gave trouble, though this is much easier through an abdominal wound. But when the mesentery corresponding to the 18 inches of resected bowel had been tied off and sutured side to side, a mass was formed of such a size that it was impossible without the exertion of more force that the sutured bowel would stand to reduce it through the femoral ring. I was therefore obliged to enlarge the opening by dividing Poupart's ligament in order to get it back. The result of this was a large femoral hernia in course of time. Taught by this, I determined in the next case to cut away the gangrenous loop after tying the bowel above and below, and then to pass the stumps, carefully covered with gauze, into the abdomen and then out again through a median incision in the abdominal wall for the performance of the extensive enterectomy (six and a half feet) which was necessary. Here, of course, there was no trouble with the mesentery in slipping it under Poupart's ligament, and the femoral opening could be closed by a few stitches. The result is that up to the present, now more than two years, there is no trace of hernia which was so marked in Case 1. This procedure, of suturing the stumps and returning the bowel after removal of the gangrenous loop and performance of the rest of the enterectomy through a median incision, appears to me the typical method of dealing with the condition.

The paper is a natural sequel to some papers on "Enterectomy for Gangrenous Hernia" published in the *Lancet* in 1903.[1]

[1] The *Lancet*, May 30 (p. 1495) and June 6 (p. 1576), 1903.

XXIII.—*A Case of Streptococcal Pachymeningitis.* By Sir Dyce Duckworth, M.D., and C. M. Hinds Howell, M.B. *Read March* 24, 1905.

W. B., æt. 14, was admitted to Matthew Ward, St. Bartholomew's Hospital, on November 18, 1904, with the following history:

He had been ailing since November 1, when he complained of headache, and had vomited after his food.

He improved, but on November 7, when first seen in the Out-Patient Department, there was stiffness of the neck, tenderness over the left sterno-mastoid, and the head was drawn slightly down towards the left side.

The boy came again to hospital on November 18. The stiffness of the neck had continued, and the temperature, normal before, was now 102° F., the pulse-rate being 90. The urine contained a slight trace of albumen. He was admitted. The past history was unimportant, but he was said to have suffered for the past two years from headache, to have been very irritable, and at times strange in his manner. He had complained of pain in the left ear for four weeks previous to admission; this pain had been aggravated by bathing.

On admission the patient was noticed to be a thin, dark-haired boy—pale, but with a bright flush on each cheek. He was extremely irritable, complained of pain behind the left ear and beneath the angle of the left jaw. There was, however, no tenderness on pressure over the mastoid region, and the membrana tympani was natural. There was photophobia to a small extent. The pupils were of moderate size, and equal; they reacted to light and accommodation. There was no squint. The fundus oculi was natural. The chest was poorly covered. There was an occasional short, dry cough, but the lungs were found to be natural. The heart's apex was in the 5th space in the nipple line; the area of cardiac dulness was increased to mid-sternum; the first sound was prolonged at the apex and the second accentuated at the aortic base. The pulse was infrequent—80 beats per minute—but regular in force and frequency. Temperature, 102° F. in the morning, was 104° F. at night. The knee-jerks were distinctly brisk; the plantar response was flexor; Kernig's sign was doubtful.

On November 21 the patient was still very irritable, and,

refusing all food by the mouth, was fed by the nasal tube. The urine now showed a much increased amount of albumen, with blood and casts. At 4 p.m. a very slight convulsion occurred, the head was turned to the right side, there was slight twitching about the corners of the mouth and face, after which general trembling occurred. Leucocyte count was 15,400.

On November 24 the patient had an attack of Cheyne-Stokes' breathing, which lasted about ten minutes. Definite weakness of the right side of the face was noticed then for the first time. The knee-jerks were exaggerated, and ankle clonus was present. Babinski's sign was present on both sides. Kernig's sign was now definitely obtained on both sides.

During the night of November 26 patient had three dyspnœic attacks, each lasting about five minutes. During these attacks the mouth was drawn very much to the left side. The patient now began to pass everything under him. The pulse was irregular, occasionally intermittent, and of very poor tension.

On November 27 the patient became comatose, and died at noon.

The temperature from November 18 to November 23 varied very slightly round 103° F., but fell during the last two days, and was 95·2° F. immediately before death. The pulse, which was regular at first, became very irregular, sometimes intermittent, during the last three days of the illness; only twice did the rate exceed 100 beats per minute; at all other times it varied from 60 to 80 beats per minute. The urine contained albumen throughout, and from November 20 blood and casts were present.

The *post-mortem* examination revealed the following very interesting condition: On opening the skull the dura mater externally was natural, but in the subdural space was a very large quantity of thick, greenish-yellow pus, which flowed away in a stream on removing the membrane. The pus extended over the whole of the vertex of the brain, with the exception of the extreme occipital region. The under-surface of the dura mater was thickened, and showed evidence of hæmorrhagic and purulent inflammation. The cerebellum above and below was quite free, nor was any pus found at the base or post-basal region. The spinal cord was free from pus. The surface of each hemisphere had been pressed by the pus into deep furrows, which ran in the sagittal diameter from the frontal to the occipital lobe, the largest being about 2 inches

wide by 1 inch deep. On washing the surface with water the pus came away, leaving a fairly normal pia-arachnoid covering the brain-tissue. On section of the brain, the extreme tip of each frontal lobe was found to be the seat of a small abscess. There was a small abscess in the lower lobe of the right lung, pyæmic in origin, the size of a walnut. The kidneys were swollen and pale, and the capsule stripped easily, the condition being probably due to a subacute parenchymatous nephritis. The microscopical examination of the pus from the meninges showed streptococci.

The case presents several interesting points apart from the very unusual local pathological condition. The duration of the illness, which must have been at least twenty-seven days, was unusually long for a case of purulent vertical meningitis, and it was most remarkable that the patient should have continued up and about for a fortnight of that time. A further remarkable fact was that no focus could be found as a primary source of infection. The absence of convulsions was curious; as the vertex of the brain was so greatly affected, these might have been expected. Beyond the slight paresis of the right side of the face, no paralysis was noted, and, save for the exaggerated knee-jerk, ankle clonus, and Babinski's sign in both feet, there was nothing to indicate the extensive compression of the motor areas which must have taken place. The pulse-rate throughout was remarkably slow, when the high temperature and the purulent character of the meningitis are considered.

Lumbar puncture was not performed, but from the subsequent inspection it is very doubtful if it would have afforded any useful information.

I have to thank Sir Dyce Duckworth for permission to report this case, which was under his care in the hospital.

XXIV.—*An Affection of the Knee possibly due to Malaria.*

By Howard Marsh, M.A., M.C., F.R.C.S. *Read April* 14, 1905.

A GENTLEMAN, æt. 38, a patient of Dr. Tatham, of Cheltenham, was kicked on the knee by a horse in December, 1902, but soon recovered. In April, 1903, the knee, for three weeks, was the seat of such severe pain that he could not sleep, but there was no swelling or other objective symptom. A few months later pain became so intense that hypodermic injections of morphia were used. The joint, however, remained cool, movable, and very little swollen. A splint was applied, and he went about on crutches. He had some hot-air baths, but the pain became still worse, and the joint was swollen, and measured two inches more than the other. The swelling, however, soon subsided, and when I first saw him, early in December 1903, the joint, on inspection, appeared natural. There was no swelling, and passive movements were perfectly free and smooth. But the muscles were very much wasted and so powerless that he could not raise his heel from the surface of the bed, and he carried a hooked stick with which he lifted the foot when, while sitting, he wished to alter the position of the limb. He complained of agonising pain from his hip to his ankle, and said it was like being on the rack. I sent him to a surgical home for further observation, and next morning I was told by the night nurse that he had had scarcely any sleep. He complained bitterly of pain, and said that something must be done. He was even ready to submit to immediate amputation. During my visit he stated that the knee became swollen every evening. On seeing him at 6 p.m. I found that the joint had undergone a very remarkable change since the morning. It now measured 1½ inch more than the other, and the swelling was such as I had never before met with. It followed exactly the limits of the synovial membrane, and within this area it was so abrupt and prominent that the joint had a remarkable globular appearance, and the patella was pushed forward in a very striking manner. There was no fluid in the joint, and the swelling seemed due to vascular turgescence of the synovial membrane, which appeared to be half an inch thick. The patient stated that the joint swelled up in this manner every evening, and then gradually returned to its natural size. Hearing of this quite definite periodicity, and having myself seen the differ-

ence in the joint between the morning and evening, I thought of ague. I found that during a long residence abroad he had had malarial poison, and had often taken quinine; he had taken none, however, since the knee had been troubling him. I gave him ten grains of quinine twice a day. Next morning I learnt that his pain was gone, and that he had slept soundly all night and until 10 a.m. He looked like another man; he was fresh and bright, and said it was the first night without pain which he had had for eight months. He continued the quinine. The pain did not return; in four days the attacks of swelling had disappeared, and he recovered muscular power in the limb so rapidly that in three weeks he was able to walk three miles easily, without any bad result. From that date, although the knee for some time remained slightly enlarged, the periodic swellings never returned. But six months later he had what he described as intense burning and shooting pain, worse at night, and extending from the crest of the ilium to the lower part of the leg. About this time a patch of subcutaneous swelling, covered by over-warm and intensely hyperæsthetic skin, appeared just above the internal malleolus, which prevented him from lacing his boot. The swelling was, Dr. Tatham found, much more pronounced and much more painful at night. The pain was relieved by phenacetin. For some time this swelling remained, and was very painful at night, although during the day the patient hunted and even followed the hounds on foot for twelve or fourteen miles across country. The continuous current was tried, but he declared it increased his pain to an unbearable degree. Then quinine was given in ten-grain doses, with the result that pain was immediately relieved, and the swelling gradually diminished.

At the present time Dr. Tatham reports that the knee is perfectly well and as strong as the other, and the patient is entirely free from pain. Last October he complained of pain in the lower part of the tibia, and a skiagram showed that the lowermost third of the bone was enlarged. There was a history which raised a suspicion of syphilis. The pain was relieved by a combination of potassium iodide and quinine.

Such are the main features of this case. In offering the following remarks I am aware that much that rests on very slight evidence, much indeed that is fanciful and erroneous, has been written on the subject of what has been termed masked ague. Moreover, as to malaria itself, whether in its characteristic forms or its more obscure manifestations, I have practically no personal experience. I ought at once to state

that the blood was examined by Major Ronald Ross, but with a negative result. I speak, therefore, needless to say, with diffidence and as one who is seeking information.

Two phenomena present themselves for discussion—the swelling of the knee and of the subcutaneous tissue of the leg; and the severe pain in the limb, with which was associated great muscular wasting, and powerlessness which almost amounted to definite paralysis. The swelling of the knee was periodic, recurring at intervals of twenty-four hours. When, however, the patient, who had previously often had malaria, had taken twenty grains of quinine, the swelling ceased to recur and it has never returned. The patch of swelling of the subcutaneous tissue of the leg also appeared to show periodicity, in that it increased at night and was then attended with intense pain, local heat, and extreme hyperæsthesia of the superjacent skin, while during the day it gave the patient so little trouble that he could run across country for twelve or fourteen miles without inconvenience.

While the patient was taking quinine not only was pain relieved, but the subcutaneous swelling itself gradually disappeared. Later on, when it had returned it disappeared again while the patient was taking quinine, now, however, combined with potassium iodide—ordered for the enlargement of his tibia, which was probably of syphilitic origin.

Is it possible that these periodic swellings of the knee and the subcutaneous tissue were due to malaria? Were they cases similar to those in which patients suffering from malaria have been attacked with coma?—to such a case, for example, as the following, recorded by Dr. Hilton Fagge: "A gentleman awoke about 4 a.m. with chilliness, nausea, and headache. After an hour he became extremely hot, the pain in his head was intense, and he passed from a drowsy condition into one of complete coma, with deep snoring, as if in a severe apoplectic fit. He seemed to derive much benefit from bleeding and other treatment, and in the morning appeared perfectly well. The day but one after the same symptoms returned and were met by the same treatment, but when a third attack came on Graves saw that it was an attack of *tertiana soporosa*, and cut it short by large doses of quinine."

Certainly so far as the observed phenomena are concerned a strong similarity can be traced. Furthermore, as bearing on the question of the local manifestations of malaria, I may relate two cases of which I have read, although I regret that I do not remember where they are recorded. In a malarial

district two soldiers underwent amputation through the thigh. In both, for several days, the stump each day became painful, engorged, and dusky, and then dripped blood freely, so that the patients' lives were in danger. Then large doses of quinine were given. The result in both cases was immediate. The swelling ceased, no further bleeding occurred, and the patients both recovered. Now, it is not going beyond matters of fact to say that, from the point of view of mere clinical symptoms, in these cases a very strong resemblance presents itself. All were examples of localised vascular engorgement, periodic in its recurrence, and at once controlled by quinine.

The pain which Dr. Tatham's patient suffered was not only periodic but intense and agonising, and it was immediately relieved by quinine; and in these features it resembled brow ague and other forms of neuralgia which are, I gather, generally attributed to malaria. The condition of the muscles of the limb claims a word of notice. The wasting and loss of power were both extreme. Certainly the manner in which power was regained when quinine was given was remarkable. Three weeks after the patient was unable to raise his heel from the surface of the bed, and was lifting his practically paralysed limb with a hooked stick, he was able to walk three miles without difficulty or fatigue. It seems obvious that some condition which had profoundly interfered with innervation had been abruptly removed by the action of quinine.

XXV.—*Cases of Intermittent Hydrops of the Joints.*

By Howard Marsh, M.A., M.C., F.R.C.S. *Read April* 14, 1905.

Case 1.—An unmarried woman, æt. 28, was admitted into St. Bartholomew's Hospital in 1903 with the history that, for eighteen months, her right knee became swollen and painful every fourteen days. The swelling, which recurred with strict regularity or periodicity, lasted for three days, and then disappeared, leaving the joint in what seemed to be a perfectly normal condition. While under observation for six weeks the patient had three attacks. Two were punctual to the day, while one, which was due on a Saturday, occurred on the Sunday—one day late. The effusion reached its acme on the second day, and then subsided, so that on the fourth day it had disappeared. The attacks were all exactly alike. The joint grew uncomfortable and stiff, the synovial cavity became somewhat tensely distended, and the superjacent skin a little over-warm. Some fluid was removed and examined. It was reported to be merely synovial, diluted with serum. Various means were tried, without benefit, but after taking small doses of arsenic, with occasional interruptions, for six months, the patient wrote (January, 1904) saying the attacks had ceased.

Case 2.—A gentleman, æt. 42, seen in May, 1904, found, at the end of 1902, that both his knees were often swollen. The attacks lasted three or four days. Between the attacks they appeared to be natural. He had noticed that the swelling seemed to return at regular intervals of a fortnight. I found both knees moderately distended (containing perhaps three ounces of fluid). They were quite cool, painless, and freely movable. I prescribed arsenic. In January, 1905, he sent me a time-table which he had kept. This showed that between May and December, 1904, he had fourteen attacks, occurring fortnightly, though not with exact punctuality; for while the great majority fell strictly on the fourteenth day, one occurred on the thirteenth day, two on the seventeenth, and one as late as the eighteenth day. Between September and December the attacks were shorter in duration and separated by longer intervals, and when the patient wrote at the end of January, 1905, none had appeared for six weeks. He had taken arsenic all along, but, as I gathered, not very regularly.

Case 3.—A schoolboy, æt. 16, had effusion into his right knee-joint (on the first occasion after a wrench at football) which returned periodically every twelfth day for a period of six months before I saw him in July, 1904. I examined the knee during an attack. As in the other cases, the synovial cavity was loosely distended, and he complained of resulting discomfort. Movement was unimpaired. The attacks disappeared in three or four days. Arsenic was ordered. His mother wrote in December to say that the attacks had grown shorter and less frequent, and had ceased in November.

Remarks.—These three cases were, in their essential features, very similar. In two of them effusion occurred into one knee-joint—in one case both knees were affected. The effusion returned with remarkable periodicity—in two every fortnight, in one every twelfth day. I say remarkable periodicity because, though the recurrence in one case was on one occasion a day earlier, and on three or four others two or three days late, yet, in a total of about fourteen recurrences, only four were delayed by as much as three days, and this when—probably, I think, under the influence of arsenic—the affection was tending to disappear. In all, the local condition —mere increase of synovial fluid—was the same. In all, the joints in the intervals appeared normal—at most the patients complained of slight weakness. As to treatment, I can only say that long courses of arsenic alone seemed to be of use.

These three cases may be regarded as typical of the usual form of intermittent hydrops of the joints. In their general features, and especially in their periodicity, they closely correspond with those observed or collected by Dr. E. G. Brackett and Dr. F. J. Cotton, and published in a valuable paper in the *Boston Medical and Surgical Journal.** In the examples therein recorded, amongst them being one published as early as 1867, in the *Transactions of the Medico-Chirurgical Society*, by the late Mr. Charles Moore, while the periodicity was always marked, the intervals between the attacks varied in different individuals. In some the interval was three or four days, in some nine or ten days; in some it was as long as thirty days or more. The interval most frequently observed was fourteen days. In some instances the interval changed, so that, whereas at one time it was thirty days, it afterwards became eight days (Moore's case). In another it changed from fourteen days to two days. The whole duration of the affection differed considerably in different instances. But in

* Vol. cxlv, p. 484 (1901).

several it extended over three, four, or more years. In several cases the attacks, after persisting for many months, ceased for some weeks or months and then reappeared—a circumstance that should be borne in mind, both when prognosis is being considered, and conclusions are being drawn as to the value of any particular form of treatment. Some cases, after showing periodicity in the most definite manner, may partially lose this feature, and the attacks may become irregular, both as to the time of their recurrence and their severity and duration.

I must express my entire ignorance of the pathology of the affection. I do not know on what the periodicity depends, nor even whether it is an essential phenomenon. It may be that, under the influence of some cause not yet recognised, a vaso-motor disturbance is produced that leads to synovial effusions and which, though sometimes, strange to say, is periodic, is not always or necessarily so. It seems possible that some cases of relapsing synovial effusion for which no cause is apparent may be of this nature, even though they are not periodic. If so, there is the danger that they may be treated on the supposition that the recurring effusion is due to a loose semilunar cartilage or an irritable synovial fringe, and the joint may be opened to no purpose; or they may be regarded as tuberculous, and a long period of rest and fixation may be ordered when such treatment is quite unnecessary.

XXVI.—*Two Cases of Hydronephrosis due to Movable Kidney.* By F. J. Steward. *Read April* 14, 1905.

SLIGHT degrees of dilatation of the renal pelvis are well known to be not infrequently associated with movable kidney, the condition being often noticed by the surgeon during the operation of nephropexy.

This dilatation of the pelvis is commonly associated with attacks of pain in the affected kidney, and is no doubt due to the fact that the sagging down of the kidney results at times in the production of a more or less acute bend in the upper portion of the ureter, thus causing obstruction to the outflow of urine.

This was described as long ago as 1856 by Gigon, who showed how the ureter was kept fixed by the peritoneum so that displacement of the kidney in a downward direction must result in the formation of a curve or kink in the ureter, and consequent obstruction. Dilatation of the renal pelvis produced in this manner may, however, result in the formation of a definite hydronephrosis. This may be brought about in two ways.

In the first place, owing to the dilatation of the different parts of the pelvis being unequal in degree, the mode of origin of the ureter from the pelvis may undergo a change. Usually there appears to be a tendency for the lower part of the renal pelvis to dilate more rapidly than the upper, so that the orifice of the ureter becomes more and more oblique, or even separated from the cavity of the pelvis by a vertical spur. In this manner the obstruction to the outflow of urine is increased and so a hydronephrosis is produced.

This condition has been frequently seen, and of late a number of cases have been cured by plastic operations upon the upper part of the ureter.

The second way in which a large hydronephrosis may be caused by a movable kidney is by persistence or aggravation of the ureteral kink caused by the sagging down of the kidney. This condition would appear to be a rare one. Mr. Henry Morris gives a tabulated analysis of the causation in 142 cases of large hydronephrosis, and in none of these cases was the hydronephrosis caused in the manner under discussion. Again, in speaking of the connection of hydronephrosis and movable kidney Mr. Morris says:

"I am much disposed to think that in several cases in

which hydronephrosis and movable kidney co-exist the mobility was acquired after, not before, the hydronephrosis, and is a result of the dragging weight of the distended organ."

Because of this seeming rarity I have ventured to bring to your notice the two following cases, in each of which there was a large hydronephrosis which was caused solely by kinking of the ureter. The clinical histories of the cases are as follows:

Case 1.—Elsie N., æt. 20, a healthy-looking, well-nourished young woman, was admitted into Guy's Hospital, under Dr. Perry, on February 20, 1900, with the following history:

Eight weeks before admission pain in the right loin first commenced. This pain, which varied in intensity from time to time, being sometimes of a dull dragging character and at other times very severe, was worse while standing or walking, but was relieved by lying down. With temporary periods of relief the pain continued up to the time of admission to the hospital. Sometimes pain on micturition was noticed, and on two occasions blood was noticed in the urine after severe attacks of pain. The patient had an attack of influenza shortly after the pain commenced, and Dr. Poole, of Wimbledon, who attended her, discovered the presence of a tumour in the right loin.

On admission a large fluctuating tumour was found occupying the right loin and extending from a point just below the costal margin to a point two inches below the umbilicus. The tumour was smooth, easily defined, devoid of tenderness, and very freely movable, so that it could be pushed under the ribs, down into the iliac fossa, and forward beyond the umbilicus. It filled the loin behind and extended forward to within an inch of the umbilicus.

The urine was of rather low sp. gr., 1009, but in other respects was normal. The amount of urea excreted was estimated daily, and was found to vary between 105 grains and 220 grains, the average during thirteen days being 167 grains.

The tumour was found after admission to vary considerably both in size and tenseness, but on no occasion did it disappear altogether. The pain was also noticed to be greater when the tumour was tense.

I saw the patient with Dr. Perry, and decided to explore the tumour.

Operation, March 12, 1900.—The tumour was exposed through an oblique lumbar incision, and was found to be a

large hydronephrosis. The kidney itself was so distended, in addition to the pelvis, that it seemed doubtful whether any secreting structure remained.

On examining the upper part of the ureter, the cause of the hydronephrosis was at once apparent, for at a point about an inch below its origin from the pelvis it was kinked, the upper portion, which was widely dilated, being sharply bent forwards and inwards upon the lower part, which was of the normal size. On pushing the kidney upwards, moreover, it was seen at once that the ureter was straightened and that the kink disappeared, a constriction remaining, however, at the junction of the dilated portion of the ureter with the part below.

The origin of the ureter from the renal pelvis was found to be normal and not in any way displaced upwards, as has been found in some cases for which plastic operations have been performed.

As the cause of the hydronephrosis was clearly the mobility of the kidney, I fixed the latter firmly to the muscles of the loin above and below the wound by means of a number of silk sutures, part of the wound being left open and lightly packed with iodoform gauze in case it should be found necessary later to drain the kidney. The wound healed well, and from the time of the operation all pain disappeared; but although the tumour was thought to be somewhat smaller, it certainly did not diminish in size to a marked extent while the patient remained in hospital. A distinct alteration took place, however, in the amount of urea excreted, for whereas the average amount *per diem* during thirteen days prior to the operation was only 167 grains, the average for eighteen days after the operation was 277 grains *per diem*, or an increase of 65 per cent. Three months after the operation the tumour was smaller, but still of a considerable size. There had been no return of severe pain, but a certain amount of discomfort was complained of.

The patient was seen again about a year after the operation; the tumour was then decidedly smaller, and practically no pain or discomfort was experienced. I have not had an opportunity of examining the patient since this, but on December 13, 1904—that is, nearly five years after the operation—I received a letter from her in which she says: "I am glad to say my side is quite well." Only yesterday, however, I had a letter from Dr. Poole, who examined her on April 11, and found that the kidney is again distended and that the tumour is, in fact, larger, he thinks, than before; but he does

not state whether the pain has returned. I presume, therefore, that the kidney has again become movable and that the ureter has again become kinked.

Case 2.—Miss E., æt. 18, was admitted under my care into Guy's Hospital on September 15, 1904.

Sixteen weeks previously a hydronephrosis on the right side had been opened and drained by another surgeon, and since the operation the wound had been kept patent by means of a tube.

On examination it was at once noticeable that, owing to the tallness of the patient, the ileo-costal space was certainly abnormally long. An oblique incision had been made some two inches below the costal margin so that the kidney clearly occupied a position considerably below the normal. The kidney, however, could not be felt.

Large quantities of fluid continually drained from the tube, and on analysis it was found to have a specific gravity of 1004, to be neutral in reaction, to be free from albumen, and to contain one grain of urea to the ounce. On temporarily removing the tube the sac of the hydronephrosis rapidly became distended and a large tumour formed. It was then seen that the kidney was considerably below its normal position, for its upper margin only just reached the costal margin, whilst the lower end lay in the iliac fossa. The tumour was also movable, but the mobility was somewhat restricted by the attachment to the scar.

On September 15 I explored the kidney through an oblique lumbar incision. On freeing the kidney it was found that the pelvis was greatly dilated, but that the kidney was comparatively healthy, although also to some extent dilated. On exposing the upper part of the ureter a sharp kink was found immediately below its origin, the convexity of the curve being directed upwards and inwards, as in the first case, and the two arms of the curve being to a slight extent adherent to one another. These adhesions were easily separated, and it was then seen that on pressing the kidney up into its normal position beneath the ribs the bend was straightened out.

I now passed a catheter through the old opening in the convex border of the kidney into the pelvis and down the ureter, which was found to be quite free from any further obstruction. I therefore fixed the kidney and the upper movable part of the ureter to the parieties by a number of silk sutures, as in Case 1. I, however, left the catheter *in situ*

in the ureter, closing the remainder of the wound round it. During the next twenty-four hours a large quantity of urine drained through the catheter, and at the end of that time the dressings were changed and the catheter removed. From this time there was no further discharge of urine from the wound, which was firmly healed ten days after the operation. The operation completely relieved the ureteral obstruction, for the kidney never became distended again.

I last saw and examined the patient a month ago; the kidney could then just be felt and was found to be firmly fixed in its normal position; it was quite free from pain and tenderness and was normal in size.

XXVII.—*A Case of Typhoid Fever with Lobar Pneumonia due to the Bacillus typhosus.* By SIDNEY PHILLIPS, M.D., and B. H. Spilsbury. *Read May* 12, 1905.

With Specimen and Microscopic Section of Affected Lung.

J. R., æt. 20, gasfitter, was taken ill on November 10, 1904, with headache, vomiting, acute pain in the back, and weakness in the legs—symptoms considered by his medical attendant to be due to influenza. He stayed in bed for a week, then returned to work, still feeling feverish and unwell and sweating freely; on November 23 there was a return of pain in the back, with some cough and scanty sputum, and he took to bed again. He gradually got worse, and came into St. Mary's Hospital under the care of Dr. Phillips on December 2.

He was then very ill, scarcely able to stand, the temperature 104·6°, respirations 42 per minute; there was tubular breathing below the right clavicle, and absolute percussion dulness with loud bronchial breathing over the lower lobe of the left lung; numerous coarse râles could be heard at the bases of both lungs. There was some scanty but not rusty sputum; there was no herpes. The pulse beat, 120 a minute, was weak and compressible, and a soft systolic murmur was audible over the apex of the heart, which seemed a little dilated; the tongue was coated, the abdomen seemed a little swelled; he had one or two doubtful typhoid spots on the abdomen. The same evening the temperature rose to 105·6°, reduced to 104° by tepid sponging. It was certain he had severe pneumonia, and probably also typhoid fever; this probability became a certainty on the next day, when numerous fresh typhoid spots appeared; the tympanites too had increased, and there was much tenderness over the splenic area; the tongue was more coated, the respirations were 44 a minute, and the moist sounds at the base of the left lung had given place to bronchial breath sounds; the urine was free from albumen or sugar.

The temperature again rose to 105·4° in the late hours of the night of December 3. During the next three days the temperature ranged from 102° to 103°; fresh typhoid spots appeared of a dusky purple tint, the spleen became palpably enlarged; the stools were of a typical typhoid

character, and on December 5 were slightly blood-stained; the patient wandered in mind and was very restless at night; Widal's reaction was obtained. On December 6 the pneumonia at the right apex was evidently resolving, the tubular breathing there beginning to disappear and moist sounds becoming audible; the respirations too fell to 36 a minute and the temperature to 100·6°. But the general condition did not improve, the pulse-rate keeping up to 120–140, and the temperature soon rising again to 103°; the skin had the pungent dry feel common in pneumonia, and the physical signs of pneumonia over the lower lobe of the left lung persisted.

Toxic symptoms now became aggravated; the patient was deaf, continually picking at the bedclothes; he was very drowsy, but replied quite rationally to questions, and took food well throughout. On December 9 he became worse, the pulse-rate constantly 140 and much weaker, the respirations rising to 48 per minute. He had taken internally from the time of admission a mixture of quinine, perchloride of mercury, and dilute hydrochloric acid. Oxygen inhalations, alcohol, and strychnine were now freely administered, but he got gradually worse, and on December 10 the pulse became almost imperceptible, the dyspnœa increased, he became difficult to rouse, and died on December 11.

At the *post-mortem* examination, conducted by Dr. John Broadbent on December 12, there were found in the lower three feet of the ileum numerous excavated typhoid ulcers in all stages, some deep, but none perforated; there were also three or four ulcers in the ascending colon.

The spleen weighed 14 oz., and was dark and soft; the liver was enlarged and soft.

The apices of the upper lobes of both lungs were very congested and œdematous, but did not sink in water. There was some lymph on the surface of the lower lobe of the left lung, and the latter on section was seen to be in a state of red hepatisation; scattered throughout it were some small white nodules about the size of a pea. The appearance of the lung on section is well shown in the accompanying illustration. The nodules looked like tubercle, and it was not until sections were cut for microscopic examination by Mr. B. H. Spilsbury that their true nature was ascertained. Mr. Spilsbury's description is as follows:

The nodules were found to be minute abscesses. In the abscesses were large masses of organisms; in some the

organisms consisted of minute cocci which retained Gram's stain; in others there were, in addition, small numbers of bacilli, whilst in others, and these the smallest abscesses, bacilli alone were present. The bacilli had uniform characters throughout, and had the morphological appearances and staining reactions of the *Bacillus typhosus*.

Immediately surrounding the abscesses the lung tissue was very congested, and the air-cells were filled with inflammatory exudation in which mononuclear leucocytes were numerous. The remainder of the lung presented the picture of delayed resolution of a croupous pneumonia. In addition to the appearances characteristic of a red hepatisation, there was thickening of the walls of the air-cells and proliferation of the connective-tissue cells with the formation of fibroblasts. The cavities of the air-cells contained numerous large round or oval cells, each containing from one to six nuclei and with numerous pigment granules in the protoplasm, *i. e.* macrophages. Scattered through the lung, generally in close proximity to the abscesses, were minute areas suggestive of hæmorrhagic infarction.

Apart from the organism present in the abscesses, and which had passed into the tissues in the immediate neighbourhood, the consolidated lung appeared to be unusually free from organisms. No pneumococci were found, and an examination for tubercle bacilli gave a negative result.

Remarks by Dr. Sidney Phillips.—The occurrence of pneumonia in typhoid fever is by no means uncommon; it may occur at the very commencement of the disease or at any period of its course; in nearly all these cases the pneumonia is due to the pneumococcus or staphylococcus.

Pneumonia due to the Bacillus typhosus, as in this case, is very rare; indeed, very few cases are on record, and this is the first case I believe recorded in England. Chantemesse and Widal refer to two cases of bronchopneumonia and one of lobar pneumonia due to the typhoid bacillus, but give no details of these cases.

Possibly future systematic bacteriological investigation may prove such cases to be of more frequent occurrence than now thought.

Dr. Polguére in his *Thèse de Paris* (1888), "Des Infections Secondaires, leurs localisations pulmonaires au cours de la fièvre typhoide et de la Pneumonie," deals exhaustively with the subject and records the results of numerous bacteriological investigations of pneumonia in typhoid fever.

One of these cases was that of a youth, æt. 21, who died from perforation of the intestine during typhoid fever. *Post mortem* the lower lobe of the right lung was in a state of "splenification," containing enormous numbers of the typhoid bacillus.

The recorded cases of true typho-pneumonia being, as far as I know, so few, generalisation on their clinical aspects seems to be scarcely justified.

Dr. Polguére, however, states that they are characterised by coming on early in the course of the typhoid attack, by very rapid development, and by a marked tardiness in resolution. My own case conformed with these observations. The pneumonia of the left lung was certainly of early onset and of rapid development, having involved the whole of the lower lobe of the left lung, when the patient came into hospital, which was presumably about the ninth or tenth day of the disease, seeing that the eruption was just beginning to appear.

The tardiness in resolution was even more marked. At the height of the pneumonia the symptoms and physical signs differed in no way from those of ordinary lobar pneumonia. There was great dyspnœa, much cyanosis, and very high fever, with loud tubular breathing over the affected area of lung. But it was very noticeable that when in due course the other symptoms of typhoid fever commenced to decline in severity, those of the pneumonia did not; indeed, they became rather aggravated, so much so that I suspected at the time that some tubercular affection had supervened upon the pneumonia.

In its general aspects the case throughout more resembled a case of pneumonia than one of typhoid fever, the pulmonary symptoms predominating over those more strictly characteristic of typhoid.

I should like to add that the microscopic slides of the lung now exhibited are the work of Mr. Spilsbury.

PLATE VI.

Illustrating Dr. Sidney Phillips' and Mr. B. H. Spilsbury's case of Typhoid Fever with Lobar Pneumonia due to the *Bacillus typhosus*.

Bale & Danielsson Ltd. print. et lith.

XXVIII.—(1) *The Operative Treatment of Certain Cases of Fracture of the Astragalus;* (2) *A Series of Cases of Fracture of the Astragalus and Os Calcis.*

By J. HUTCHINSON, Jun., and HUGH LETT. *Read May* 12, 1905.

A RECENT French writer, M. L'Ombrédanne,* concludes a lengthy review of the subject of "Fractures of the Astragalus" by the following statement: "It is now beyond dispute that all cases of fracture of the body or neck of the astragalus are best treated by excision of that bone. If treated otherwise these fractures end, if not in anchylosis, at least in absolute inability of the patient to walk. On the other hand, the results of excision of the astragalus are absolutely perfect. . . ."

This statement is far too sweeping. Probably most surgeons will agree that the cases of astragalar fracture requiring open operation form the exception and not the rule. The following case is narrated for two reasons—first, as an instance of the necessity for operation owing to the complete failure of conservative measures; and secondly, as proving the difficulty of obtaining good use of the foot even after excision of the bone. To secure an "absolutely perfect" result requires prolonged and careful after-treatment, a point worthy of emphasis.

Both the diagnosis and treatment of fractures of the astragalus have been immensely helped by the introduction of the X rays. In fact, it is only since radiography was discovered that we have been enabled to form a true estimate of the frequency of fracture of the two main tarsal bones, to make a correct diagnosis in such cases at an early moment, and to decide on the best form of treatment to adopt. In the case to be immediately narrated the X-rays were of the greatest value. The patient was examined by several surgeons, who agreed only in the impossibility of deciding on the exact cause of the deformity without the evidence obtained by skiagraphy.

* L'Ombrédanne, "Fractures de l'Astragale," *Revue de Chirurgie*, vol. xxvi, 1902, pp. 414—435.

Walter P., æt. 14. Admitted to the London Hospital under one of us (J. H.), October 18, 1903. He slid 80 feet down inside a canvas fire escape; no one was holding it, and he consequently descended with great violence on to a grass lawn, feet foremost. His left heel must have received most of the force of impact, and probably, the toes being caught by the canvas, the front part of the foot was hyper-extended. He was unable to move the ankle at all, but had no pain until ten minutes later, when it came on acutely. From the time of the accident he was quite unable to walk with the injured foot to the ground. On admission there was much swelling and discoloration round the ankle, and considerable pain on attempting to flex the foot dorsally. Some ten days had elapsed between the accident and his admission to the hospital. No diagnosis except that of severe sprain had been hitherto made. The foot was considerably inverted and extended, and the patient was unable to flex the ankle or to evert the foot, the inversion and extension being a fixed condition even under an anæsthetic. There was hardly any pain except on attempted flexion. The external malleolus was further back than normal with relation to the tendo achillis and the os calcis, and there was an abnormal projection of the astragalus immediately in front of it. On the inner side there was an abnormal hollow below and in front of the internal malleolus.

Skiagraphs showed that the antero-posterior width of the astragalus was increased; laterally the neck of the astragalus and the os calcis were seen to be uninjured, but the space between the os calcis and tibia was diminished. Evidently there was a fracture of the astragalus (of the body rather than the neck) by the impact of the os calcis, which had compressed the bone between it and the upper bones of the ankle, the latter holding it like a vice.

It was clear that the deformity of the foot was entirely due to a comminuted and impacted fracture of the astragalus alone, one in which the impaction was greatest on the lower aspect so as to give a downward tilt to the head and neck of the bone. To allow this impaction to persist would be to leave a crippled and malformed foot, but as the osseous tissue in a subject only 14 years of age is considerably softer than that of an adult, we expected that forcible manipulation would much improve the condition. Accordingly, under full anæsthesia, the foot was strongly wrenched by the surgeon's hands. Crepitus was distinctly felt, and as flexion at the ankle

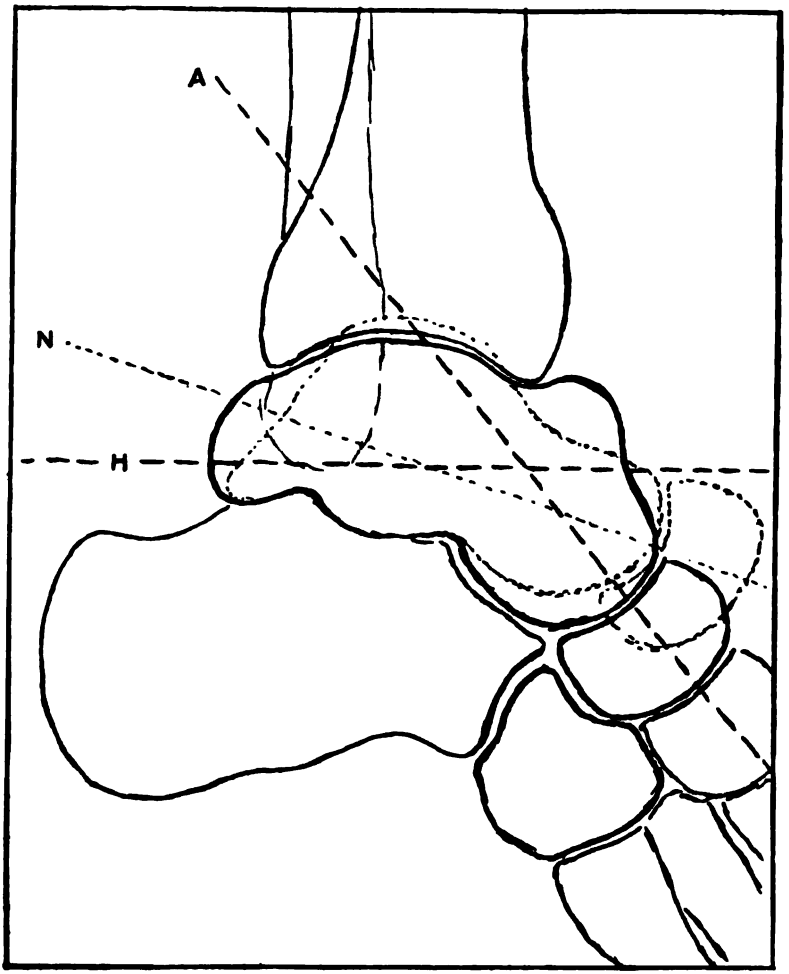

FIG. 1.—From radiograph of case of impacted fracture of the astragalus, the body of which is vertically compressed, at the same time the neck is bent downwards, and with it the front part of the foot. H, a horizontal line. N, the axis of the normal astragalar neck (in dotted outline), forming an angle of 10° with the horizon. A, the axis of the displaced astragalar neck, forming an angle of 45° with H.

became somewhat easier, the foot and leg were carefully adjusted on back-and-side splints. Daily massage was carried out over the muscles of the leg, with frequent readjustment of the splints. At the end of three weeks, however, it became evident that the result would be extremely poor unless an operation were done; in other words, the inversion and extension persisted to such an extent that the patient would be unable to walk with his foot flat on the ground. (See Fig. 1).

On November 28, about seven weeks after the accident, the operation was performed. An external incision was made between the extensor and peronei tendons. By drawing the former aside it was easy to expose the astragalus, practically the whole of which was removed in one piece. The interosseous ligament between the astragalus and os calcis had not been torn (or only slightly so) and required division with the knife. The only part of the bone left consisted in the articular surface of the head. It was then easy to place the foot in perfect position at right angles with the leg. Union by first intention resulted.

It was found that strong impaction had occurred in a line running obliquely downwards and outwards, so that the head and neck of the bone were placed at a lower plane and bent downwards; in addition the tibial surface of the astragalus faced forwards and outwards. The specimen proved that no manipulation could possibly have succeeded in removing the deformity. The foot being in such excellent position and a clear space remaining between the tibia and os calcis it was confidently expected that no difficulty would occur in obtaining a movable and useful foot. But now arose one of the most interesting points in the case. The foot was kept on a splint only long enough for the wound to heal soundly, massage and passive motion being practised within a fortnight. The greatest difficulty was experienced, however, in preventing the new "ankle" stiffening in an awkward position and in securing a proper way of walking. For many weeks, in spite of daily massage and manipulation, the boy could only bring the toes to the ground in walking. It was only after two months' practice and training that ultimately an excellent result was obtained. The foot is now strong and steady and the gait almost perfect, aided by a thicker sole on the corresponding boot.

M. Monod * treated in the Hôpital Saint-Antoine in

* Quoted by Ombrédanne, *Revue de Chirurgie*, vol. xxvi, p. 420.

Paris a case almost precisely similar to the one just narrated. A man, æt. 27, fell on to his left heel a distance of eighteen feet. The swelling of the foot was enormous, the foot being displaced inwards (varus position).

The diagnosis was made of bi-malleolar fracture, a radiograph apparently not being taken at this time. Under chloroform reduction was attempted of the deformity, and the foot and leg were fixed in plaster of Paris. Six months later the patient was again admitted into hospital since he could not walk or stand. The inversion and extension and the deformity persisted as before; below the external malleolus was a deep depression, at the bottom of which the finger detected a hard and irregular surface. Below the internal malleolus was an abnormal bony lump, over which the tendon of the tibialis anticus was tightly stretched. Both flexion and extension of the ankle were limited and painful. The radiograph showed a vertical compression of the astragalus, involving the body only of this bone. An operation was performed, the whole bone being removed in segments. It was found that an oblique fracture had passed inwards and backwards, that the upper articular surface faced outwards and forwards, while the neck was twisted inwards. The whole bone had been sub-luxated. It will be noted that in M. Monod's case the direction of the fracture was not identical with that in ours; the persistent deformity was, however, exactly the same.

The after-result is not given in full, but it is stated that a month after the operation the foot was "in very good position." Other cases of successful excision of the astragalus after fracture have been reported by Hancock, Russell, Bergeret, Desfosses, Legueu, and others.

In most of the recorded cases, however, the fracture has been a compound one, in which the indications for operation are more obvious than in such cases as M. Monod's and our own.

We may now contrast with these certain cases of old fracture of the astragalus in which the treatment has been conservative. Of course they offer much variety in the amount of bone broken off, the direction of fracture, and its probable mechanism. But one thing is very striking, namely, the frequency with which the proper functions of the foot are permanently diminished. This unfortunate result seems to arise from various causes. In some there has been comminution or impaction of the body, and as a rule equino-varus of the worst type has persisted. A research on the

effect of reduction in displaced Colles' fracture which has been carried out lately in the London Hospital leads to the conclusion that in many cases the reduction is only partial. In other words, the surgeon sometimes deceives himself in believing that his manipulation has removed the impaction and corrected the deformity. If this is true in Colles' fracture, where upper and lower fragments are just under the skin and can be readily grasped, how much more is it in cases of fracture of a deeply placed bone like the astragalus!

There are, however, a few instances of compression-fracture of the astragalus in which the force has acted vertically, and neither inversion nor eversion of the foot results. Such cases may probably result in stiffness of the ankle, but no deformity sufficient to justify operation. The following is an example in which the result was tested a year after the accident:

Charles K., a sailor, æt. 28, and of medium weight, jumped from a ship into a lifeboat. The height was said to be only five feet, but he seems to have alighted heavily on the left foot, which was bruised and swollen. No splints were applied, the only treatment being the use of liniments. At the end of ten days or a fortnight he began to use the foot. He experienced considerable difficulty in this, and at the end of thirteen months he came to the London Hospital complaining of heat, swelling, and pain whenever he stood or walked for more than half an hour.

On examination there is a well-marked bony swelling below and behind the internal malleolus, about $1\frac{1}{2}$ inches by $1\frac{1}{4}$ inches extending downwards and backwards towards the point of the heel. Below and behind the swelling ceases abruptly, below it is apparently limited by the sustentaculum tali, which is exactly the same distance from the sole in both feet. There is tenderness round the margins of the swelling. The head of the astragalus is felt in its normal position. There is no alteration in measurement between the head of the astragalus and the nearest point of the internal malleolus in the two feet. The long arch of the left foot is not diminished as compared with the right. Measurement from tip of internal malleolus to tip of external malleolus round the sole is $7\frac{1}{2}$ inches in both feet.

From the above examination the chief point made out was that the posterior surface of the astragalus projected abnormally, and a fracture of this part of the bone might be inferred. A radiograph taken from the front of the ankle showed nothing

definite, but one taken laterally revealed alteration in the whole body of the astragalus, which had evidently been compressed and comminuted, so that its lower surface had become convex. Behind it projected over the os calcis to within half an inch of the posterior edge of the bone. As a year had elapsed since the accident all trace of the lines of fracture had been obliterated. A good deal of the enlargement of the bone might be attributed to callus.

Flexion and extension of the ankle were perfect, but pain was felt on rotation or lateral movement of the foot. It was naturally decided that no operative treatment was justifiable, nor do we think that an early excision of the fractured bone would have given as good a result.

In a case under M. Vallas, of Lyons, the anterior border of the tibia had been driven into the astragalus and both bones had given way. The ankle had been fully flexed at the time of the injury. What made the case unique was that the lesion was symmetrical, and in both feet the astragalus was excised. The case is imperfectly reported (Destot, *Revue de Chirurgie*, vol. xxvi, pp. 224 and 231; Vallas, *ibid.*, p. 462), but one gathers that the accident had been a fall on to both feet from a height. Before the resection there had been equinus. On one side the external malleolus had been divided as well as the astragalus excised. The result (in spite of suppuration on one side) is said to have been very good, but this should be taken with reserve, as at the time the report was made the patient was only getting about with crutches, and "beginning to walk in a proper attitude."

Some writers (*e. g.* M. Destot) have exhausted their ingenuity in classifying fractures of the astragalus, enumerating some twenty or more types of different forms. It may save trouble to note that lines of fracture of this bone have been observed in every conceivable direction, inclusive even of an antero-posterior vertical split, and that no purpose is served by any such minute classification. The material on which it could be based is not large. One of us (H. L.) analysed all cases of fracture of the lower extremity treated in the London Hospital during two and a half years (January, 1901, to end of June, 1903). Out of 1034 such cases there was one undoubted fracture of the astragalus and one doubtful case, only two in all. There were, however, six cases of fracture of the os calcis.

We would suggest that for practical purposes simple fractures of the astragalus may be divided into two classes—those

in which open operation is called for, and those which are best treated by conservative measures.

(1) Where operation is indicated. Cases of impacted fracture in which marked deformity (usually of equino-varus type) is present and cannot be overcome, as in the case reported first in this paper. Cases in which the head of the bone is broken off in front of the interosseous ligament and is driven under the skin so as to irritate by acting as a foreign body.

(2) Where no operation is required. This class will include, in spite of the assertion of M. L'Ombrédanne and other writers, the majority of fractures of the astragalus.

For it may be fairly urged that this bone is of considerable value in the mechanism of the foot, that it is the keystone of the longitudinal arch, that its fractures repair well if the process of union is not interfered with by needless massage and passive motion during the first week or two, and that even a slightly deformed astragalus is superior to a gap between the tibia and os calcis.

There is no other bone in the body which the surgeon has been seriously advised to excise every time it has sustained an uncomplicated fracture.

A suggestion may here be made with regard to the cases of dislocation of the astragalus forwards and outwards. As is well known, these have sometimes proved irreducible even after tenotomy of the tendo Achillis and other tendons, and in spite of open incision over the dislocated bone. The treatment advocated under such circumstances has been excision of the astragalus, which certainly corrects the deformity, but may leave a somewhat crippled foot. To avoid this we would suggest a small incision over the external malleolus, which should be chiselled completely through at its base into the ankle-joint. It is difficult to believe that any further obstacle to the return of the dislocated astragalus would be experienced, and the fractured malleolus would unite in a few weeks' time with or without wiring.

Fractures of the os calcis.—The following series of seven cases were all verified by radiographic evidence. In four of them the final result as regards function of the foot was recorded.

Case 1.—Albert M., fell ten feet, turning a somersault on the way and alighting on one heel. Fracture of the body of the os calcis, running upwards and forwards from the heel, no

apparent displacement. It was three months before he could do much with the foot, and five before he walked well. At the end of eighteen months when we examined him the result was perfect.

Case 2.—Charles M., æt. 63, fell to the bottom of a ship's hold, some twenty feet, striking the right heel. Crepitus could be made out, but there was no apparent deformity. It was six months before the foot was really strong again. After three years had elapsed we found the posterior part of the os calcis thickened and projecting backwards abnormally. Eversion and inversion of the foot were limited, but the patient walked very well.

Case 3.—Ernest K., æt. 44, fell five feet, coming down heavily on to the right heel. The body of the os calcis was fractured. Three years later the foot was "fairly strong but very painful at times."

Case 4.—Christopher B., æt. 56, fell seven feet on to his left heel. The fracture lay behind the articular surface for the astragalus, crepitus being obtained. When seen fourteen months later there was flat-foot on both sides, some bony thickening below the left external malleolus. No difference could be noticed in his walk.

Case 5.—Patrick R., fell twelve feet. The fracture passed upwards from the tuberosities to the middle of the articular surface for the astragalus.

Case 6.—James H., æt. 59, fell on to his right heel a distance of eight feet. There was much swelling round the os calcis, with abnormal mobility between the anterior and posterior parts of the bone. The radiograph showed a slightly oblique fracture running up from the tuberosities towards the sustentaculum tali.

Case 7.—Thomas G., æt. 23, fell eighty feet. His right foot sustained the brunt of the injury, and on admission there was well-marked swelling below both malleoli, especially the internal one. The width of the os calcis was increased and abnormal bony projection felt and seen in front of the heel. The fracture was a comminuted one of the body of the os

calcis. A good recovery was made, with some flattening of the foot.

In addition to these seven cases, another, making eight in all, has recently come under observation, but it presented the features common to the majority, and therefore details of it may be omitted.

Fracture of the body of the os calcis is much more common than that of the posterior or heel extremity of the bone; it is usually comminuted and results from a fall of considerable height.

Apart from the invaluable evidence given by radiography, the diagnosis can usually be made with ease from the following considerations:

(1) There is a band of deep ecchymosis and swelling reaching from one malleolus towards the other, with marked tenderness.

(2) As this subsides bony deformity in the sense of widening of the os calcis and sinking of the arch, with irregular projection, can be made out.

(3) Whilst movements at the ankle-joint and mid-tarsal one are rarely interfered with, the astragalo-calcaneal movement (inversion and eversion) is usually impaired and painful.

(4) Occasionally crepitus is obtained, but this cannot be relied on. Recovery is slow, it being always some two or three months, sometimes more, before the patient can bear his weight well on the foot in walking. Frequently some degree of flat-foot persists. Nevertheless it is improbable that active surgical interference could effect more than rest on a well-padded splint, with massage from time to time. The ultimate functional result is satisfactory as a rule.

XXIX.—*A Case of Milky Ascites in which the Opalescence was not due to Fat.* By FREDERICK TAYLOR, M.D., and JOHN FAWCETT, M.D. *Read May* 26, 1905.

CASES of milky serous effusions in which the opacity is not due to fat are of sufficient rarity to merit a record being made of them, and it is for that reason that we venture to bring the details of the present case before the Society.

Joseph C., æt. 31, was admitted into Guy's Hospital under the care of Dr. Taylor, on October 6, 1904.

There is no fact of importance in his family history. He himself had never been abroad, and, with the exception that in October, 1903, he had a chancre, followed by an abscess in the groin, which was opened, and healed in three weeks, he had had no illnesses apart from those of childhood.

History of present illness.—In May, 1904, the patient began to complain of abdominal pain of an intermittent character, chiefly felt on the right side. He was sick on several occasions. Three weeks later he noticed that his "ankles and stomach" were swollen, and that shortly after his face became affected. He was told by a doctor that he had Bright's disease, and after staying at home for five weeks (May 26 to June 30) he was admitted to the Leicester Infirmary. Here he remained for seven weeks (July 1 to August 18) and during that time his abdomen was tapped on four occasions; and on one occasion the legs were punctured, when the fluid which exuded was seen to be clear at first, but after four days became milky. While he was lying in bed the legs did not commence to swell until the abdomen was full enough to require tapping, and then it was only the backs of the thighs which were affected.

From August 18 to October 5 he was at home, and during that period the abdomen was tapped three times.

We understand from a letter from Dr. G. T. Wrench that at the time the patient was in the Leicester Infirmary the fluid, which was milky throughout, contained fat, and had other characters as follows:

(1) On shaking up with ether and evaporating, the residue stained black with osmic acid. (2) Under the microscope no globules were seen, but only granules. (3) The appearance was that of milk diluted four or five times. (4) Filtering made no difference in the fluid.

Condition on admission into Guy's Hospital—The patient was a light-haired, pink-complexioned man. He was very thin, and the muscles generally were undersized and flabby. The ankles were slightly œdematous, but the swelling of the lower extremities was most marked in the thighs. The abdomen was much distended, measuring 37¼ inches in circumference, and physical signs of free fluid were present. Nothing abnormal was detected in the heart, lungs, or central nervous system.

Urine.—Sp. grav. 1014. A considerable quantity of albumen was present, about 14 parts per 1000, as measured by Esbach's method. Some fat was said to be present, but this is doubtful. There were a few hyaline and granular casts.

Blood-examination.—No filariæ were found. Differential count: Polymorphonuclear cells 159=63·6 per cent.; small lymphocytes 62=24·8 per cent.; large lymphocytes 21=8·4 per cent.; eosinophile cells 8=3·2 per cent.

October 12.—Physical signs of fluid were detected over the bases of the lungs.

October 17.—Paracentesis abdominis was performed, and 22 pints of a thin milky fluid were withdrawn.

From October 12 to 21 the patient had diarrhœa followed by progressive wasting.

October 28.—Paracentesis was again performed, and this time 15 pints were extracted. The clinical clerk states, in the report, that the fat present was in the form of large droplets, staining with osmic acid. Of this statement we can obtain no confirmation, and are informed by the house-physician that such large droplets had never, to his knowledge, been seen.

October 30.—Diarrhœa re-commenced. Marked anorexia. Patient became gradually weaker and died on November 8, 1904.

Urine.—Specific gravity varied from 1014 to 1022, and the urine contained from 10 to 20 parts per 1000 of albumen. Granular and hyaline casts were present. The amount passed *per diem* varied from 30 to 50 oz.

The temperature throughout was subnormal, and the pulse rate varied between 90-100 per minute.

Autopsy.—There were five oz. of milky fluid in the right pleural cavity and 58 oz. on the left. A large quantity of a similar fluid was present in the peritoneal cavity. No focus of lymphatic obstruction, no enlarged glands, and no growth could be found.

The pancreas weighed only 60 grammes—that is, some 25

grammes less than the lower limit of the normal pancreas. It was very hard, and its outer surface was more irregularly nodular on its outer aspect than is usual. On microscopical examination, however, the glandular tissue appeared to be healthy, and there was no increase in the connective tissue of the gland. The "interalveolar cell-islets" were very distinct. The kidneys were pale and swollen, the result of a tubal nephritis. Apart from the changes mentioned above there were no abnormal appearances to be seen in any organ of the body.

Chemical examination of the ascitic fluid obtained at the post-mortem *examination.*—Mr. J. H. Ryffel, B.Sc., Demonstrator of Chemistry at Guy's Hospital, very kindly examined the fluid for us, with the following result: Fat, ·045 per cent.; proteid, ·396 per cent.; iodine, ·0966 per cent., probably as KI.; chlorine, ·264 per cent., as chloride.

Mr. R. W. Allen, B.Sc., Gull student in pathology, has also given the following account of the analysis of the fluid as made by him: Colour, milky; reaction, slightly alkaline; specific gravity, 1010; albumen, 4·5 parts per 1000 by Esbach's method.

On precipitating the albumen by saturation with ammonium sulphate, the fluid became quite clear.

No fat globules could be detected under the microscope, and extraction with ether failed to show any fat; it, however, removed the proteid, and the fluid as before became perfectly clear.

No sugar nor fatty acid could be detected in the fluid. All the proteid was present as albumen, except for a very slight trace of albumose and peptone, probably due to bacterial action.

The action of the fluid upon starch and fat was tested at a temperature of 40° C., and after the addition of a small quantity of sodium carbonate; no change could be detected in 15 minutes. Mr. Allen says that the absence of any ferment may therefore reasonably be assumed.

Remarks.—The analysis, and the examination of the fluid obtained after death, show that the opaque milky appearance of it was due, not to fat, but probably to proteid in some form. On referring to the report of the case it will be noticed that previous examinations were said to have demonstrated the presence of fat, though possibly in no very large amount. The examinations of the fluid made during the life of the patient were not so complete as those made afterwards, and

in the report furnished by Dr. Wrench it is stated that "granules," and "no oil globules," were seen, and that the appearance was that of milk diluted four or five times. These two statements suggest that the amount was certainly not very large, and that the opacity was due, or partly due, to some other substance than fat.

Chylous ascitic fluids may contain from 1 to 4 per cent. of fat, whereas in this case the amount was only ·045 per cent. Having taken the above facts into consideration, we have come to the conclusion that this case should be included amongst those in which the opacity is due, not to fat, but to a proteid body.

Lactescent serous effusions in which the opacity of the fluid is due to a proteid have only been recorded in recent years. Dr. Hutchison pointed out that the milkiness of the fluid has been ascribed (1) to lecithin; (2) to mucoid substances; (3) to a proteid resembling casein. His own case he includes under the third of these varieties.

There seems at any rate to be some evidence that lecithin is of importance in connection with the causation of these milky ascitic fluids. Joachim, in a recent paper, refers to von Bernert's researches that albuminous bodies of the globulin group may have their solubility lessened either by chemical combination with lecithin or by their molecular arrangement.

Christen concludes that lecithin, whether in solution or not in ascitic fluid, is not in itself the cause of the turbidity.

Joachim's conclusion is that the milkiness is probably due to a combination of lecithin and a proteid (pseudo-globulin), and thus is confirmatory of von Bernert's researches, which he quotes in his paper.

It is recognised that a creamy or milky blood-serum may owe its appearance to a proteid, and it would have been interesting to know if such a lactescence of the serum had been present in this case, but unfortunately the blood was not examined. In the report of the case the statement is made that on one occasion, when the legs were punctured, the lymph which first flowed out was clear, but later became milky. This lactescent condition is stated by Castaigne to be frequently associated with acute and subacute nephritis. In the case we have described the cause of death was a tubal nephritis, and long ago Bright drew attention to the cases of some patients with dropsy whose blood-serum became turbid; and after it had stood for some time a layer of a creamy

substance appeared on the surface which did not contain "any proper oily matter in it."

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XXX.—*A Rhinoplastic Operation.* By James Berry, B.S., F.R.C.S. *Read May* 26, 1905.

MR. X., æt. 33, consulted me at the beginning of October, on account of syphilitic destruction of the left ala of the nose.

In December, 1903, he had acquired syphilis from a native woman in a remote part of Asia, where he was then living. He was at first in the hands of a native doctor, who apparently did not diagnose the disease. At any rate, it was not until three months later (March 10, 1904) that mercurial treatment was begun. At the end of this month his gums were slightly affected by the mercury, but for a short time only. Mercurial inunction was then substituted for the treatment by pills which had hitherto been adopted. At the end of April the nose, from which there had been a considerable amount of discharge, began to swell. In June the swelling and inflammation of the nose increased, and at the beginning of July the patient was again given mercury internally.

About this time a hole appeared on the left side of the nose at the junction of the cartilage with the nasal bone. This hole rapidly enlarged downwards, until by the middle of July the whole ala had been completely destroyed. He then came under the care of Major Pilgrim, I.M.S., of Calcutta, who put him on iodide of potassium (ten grains three times a day), while continuing the administration of mercury. Under this treatment the ulceration began to heal rapidly. A few weeks later the patient was sent back to England and advised to consult me about a plastic operation for the repair of the deformity.

I saw him for the first time on October 4. At that time his general health was excellent. The whole of the left ala of the nose had completely disappeared right up to the lower edge of the nasal bone. The tip and the rest of the exterior of the nose were quite sound and not inflamed.

As, however, there still remained a small patch of ulceration just inside the nostril, and as the inflammation had but recently subsided, it was considered best to postpone for three months any question of operation. In the meantime the patient was fitted up with a coloured artificial aluminium ala, which he wore during this period, and which proved very satisfactory.

Fully realising the difficulties of plastic operations upon the

nose, especially in syphilitic cases, I should have been quite content to have left the patient in this condition, with an artificial nose, which, from an æsthetic point of view, was certainly better than anything I could make for him by operation. But as he was about to return to Asia, where he would have no opportunity of repairing any possible damage to the artificial substitute, and where the hot climate would prevent the use of the adhesive gum by which the artificial ala was affixed, the patient insisted on my attempting to restore the nose with flesh and blood.

The patient was quite willing to submit, if necessary, to the Tagliacotian operation, but it seemed undesirable to subject him to so irksome and severe a proceeding for the restoration of so comparatively small a portion of the nose. The Indian method of rhinoplasty was obviously not suited for such a case, owing to the distance of the ala from the forehead.

To obtain the tissue necessary for the formation of a new ala, the choice, as it seemed to me, lay between taking a flap from the upper lip and taking it from some part of the cheek. The lip method was rejected mainly on the ground that moustache hair would grow on the restored ala and require daily shaving. Otherwise the thick tissues of the lip would have provided excellent material.

The method finally employed was that of utilising the tissues of the naso-labial fold for the necessary flap, care being taken to cut it in such a manner as neither to draw down the eyelid nor to distort the lip. The scar, too, had to be placed where it would be least conspicuous.

The object of the operation was not merely to lay a bridge of skin over the exposed nostril, but to place a thick solid mass of skin and muscle in the place of the lost ala. Allowance had also to be made for contraction of the flap, which therefore had to be cut much larger than it was ultimately intended to be. Fig. 1 shows the patient's condition just prior to the operation, which was performed on January 14, 1905, in the following manner: A straight incision 4·3 centimetres (nearly 1 inches) long was made down to the periosteum along the line ADB (see Fig. 1) from the left side of the tip of the nose, along the anterior edge of the hole in the nose and up to a point 6 mm. (¼ inch) below and 4 mm. internal to the inner canthus of the left eye. From the upper end of this incision a second (BEC) of about the same length was carried downwards and outwards along the upper or

outer border of the naso-labial fold, thence curving slightly downwards to a point on the cheek about 25 mm. (1 inch) external to the outer margin of the gap that had to be covered in. The flap thus obtained, which included everything down to, but *not* including, the facial artery and vein, was then dissected downwards, and the neighbouring parts of the cheek and upper lip were freely raised by undercutting. The tissues on either side of the line AD were then dissected up for about 4 mm. ($\frac{1}{6}$ inch) so as to form a deep groove for the reception of the edge of the transplanted flap.

The latter was folded upon itself and then twisted inwards and downwards, so that the upper angle (B) was brought down to the tip of the nose at (A). The upper half (BE) of the outer edge of the flap was then accurately sutured with fine fishing gut to the lower half (AD) of the first incision along the side of the nose. The raw inner edge of the flap was thus left free to form part of the anterior edge of the restored nostril. This granulation edge appears in Fig. 2 as a black area, which subsequently curled inwards and disappeared within the nostril behind the new ala. The lower part of the outer edge of the flap thus became acutely doubled upon itself, and the skin edges of the V-shaped area thus formed were accurately united by a couple of fine sutures. The flap was thus made to present a prominent angle in imitation of the prominent part of a normal ala. The line of suture last-mentioned fell into the cleft between the ala and the cheek, and was ultimately invisible.

A triangular raw area (DBE), each side of which measured about 25 mm. (1 inch), was still left at the upper part of the wound. I had expected to have to close this in part by grafting, but it was found that this was not necessary, the two lateral sides of the triangle being brought directly into apposition with sutures, after a good deal of undercutting, chiefly on the side of the cheek. There was, of course, great tension along the lower part of this line of suture. Indeed, the suturing could not be completed until a double deep-tension suture of stout fishing-gut had been passed deeply through the tissues of the cheek and nose. The ends of this suture came through the skin respectively at the centre of the nose and at a point nearly midway between the angle of the mouth and the external canthus of the eye. At these two points the ends were tied over short pieces of rubber tubing (*see* Figs. 2 and 3).

Throughout the operation bleeding was checked by

sponge pressure, no ligatures being employed. A collodion and gauze dressing was applied. The whole operation lasted 54 minutes.

The subsequent progress of the case was uneventful. No elevation of temperature or any other complication ensued. Syphilitic inflammation of the parts, which I had feared might occur, was conspicuous by its absence. Some of the stitches were removed three days after the operation and the remainder on the fifth day, excepting the deep-tension stitch, which was left undisturbed until a whole week had elapsed. In the meantime the pieces of rubber tubing were gradually cutting their way by ulceration through the skin, so that when this stitch was finally removed two small ulcers remained in the nose and cheek. These, the scars of which are visible in Fig. 3, healed in the course of a few days.

Fig. 2 shows the condition seven days after the operation just before the removal of the deep tension stitch.

On February 4 the patient left the nursing home and went down into the country, all dressings having been discontinued several days previously.

On March 10, by the kindness of our Secretaries, I was allowed to show the patient to the Society as he was passing through London on his way back to Asia. Figs. 3, 4, and 5 show the condition a few days before that date. Fig. 5 shows the under surface of the new ala, and the photograph would perhaps suggest that the nostril was completely blocked; such, however, was not the case. The nostril measured 7 mm. transversely, and the patient could breathe quite freely through it. It may be expected that as the flap shrinks the nostril will become still more widely open.

I have ventured to bring this case under the notice of the Society partly because detailed accounts of such operations are comparatively rare. Another reason for recording the case is that I believe some of the details of the operation are new, and will, I hope, prove of service to those who may have to deal with similar cases. I refer more especially to the doubling up of the flap so as to give a cutaneous surface to both surfaces of the restored ala.

A point upon which I hope to elicit opinions from those present, and one upon which I am myself doubtful, is the length of time which should be allowed to elapse in these syphilitic cases between the subsidence of acute symptoms and the performance of the plastic operation. I think it highly important for the success of such an operation that

antisyphilitic treatment by iodide of potassium and mercury should be carried on continuously before, during, and for some time after the period of operation, as was done in this case.

PLATE VII.

Illustrating Mr. James Berry's paper on A Rhinoplastic Operation.

FIG. 1.—Just before operation.

FIG. 2.—Seven days after operation; immediately before removal of the deep tension stitch.

FIGS. 3, 4, and 5.—Two months after operation; showing the restored ala and the scars.

FIG. 1.

FIG. 2.

FIG. 5.

FIG. 3.

FIG. 4.

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XXXI.—*Case of Primary Adrenal Carcinoma of the Liver.* By Sidney Phillips, M.D., and Mr. B. H. Spilsbury. *Read May* 26, 1905.

M. B., æt. 40, was admitted under Dr. Phillips' care at St. Mary's Hospital on December 15, 1904. She was the mother of nine children, six of whom were living; one had died of bronchitis, one of diarrhœa; she had had one miscarriage. She had had good health, and had been in the habit of taking about one pint of beer daily and sometimes a little brandy. Her father, she stated, suffered from heart disease; her mother had died of "abscess of the liver."

Throughout the year 1904 she suffered from loss of appetite and indigestion, feeling sick at the sight of food and sometimes vomiting; her doctor told her she had an affection of the liver. She gradually lost flesh and strength, and early in November took to her bed. She then for the first time noticed enlargement of the abdomen, which gradually increased. About the beginning of December she began to have pain in the umbilical region.

On admission into the hospital she was very sallow and emaciated, complained of pain in the epigastrium after food and abdominal discomfort. There was great distension from ascites, and 386 oz. of clear liquid were withdrawn by a trochar; this gave her much relief and enabled the liver to be felt, enormously enlarged. The left lobe could be felt to be a mass of nodules of all sizes up to a pigeon's egg and with umbilicated surface. The right lobe felt less universally nodulated. The fissure in the liver could be readily felt, but not the gall-bladder.

Below the liver and apparently attached to the colon there could be felt a rather hard nodule the size of a tangerine orange, and there were scores of small nodules feeling like lymphatic glands, floating as it were in the abdominal cavity, some just under the abdominal wall, evidently nodules of growth in the peritoneum.

There was no jaundice and no pyrexia, but the patient was very weak and emaciated, and died of exhaustion on December 26.

The *post-mortem* examination, conducted by Dr. Broadbent on December 27, showed the peritoneum and the walls of the intestines to be studded by nodules of growth of small size,

mostly about the size of a pea or a bean; some were merely cysts containing transparent, clear, yellow liquid; some were on the visceral, some on the parietal peritoneum.

The right lobe of the liver was very much enlarged, with numerous nodules of white colour scattered through its substance and on its surfaces. On the surface of the left lobe there was hardly any spot of liver substance uninvolved by nodules, the convexity and edges of the liver being little more than a mass of confluent nodules. On section of this lobe of the liver there were extensive tracts of homogeneous white appearance, consisting of growth, with islets of the same scattered through the remaining liver area. There were no gall-stones in the biliary passages. A hard mass, the size of a walnut, laid in the angle between the ascending and transverse colon, and partly surrounding the latter; this could be dissected away from the intestine without injuring the latter. The head of the pancreas had on it a small mass of similar growth. The great omentum was also a mass of growth. The supra-renal bodies and all other organs of the body were quite healthy.

The case, as seen in the *post-mortem* room, was not apparently different from other cases of carcinoma of the liver and peritoneum, and it was not until sections were cut by Mr. Spilsbury that its true nature was ascertained.

Mr. Spilsbury's description is as follows:

On microscopical examination the tumours presented the characteristics of a carcinoma originating in the supra-renal cortex. The secondary nodules in the right lobe of the liver, having the most typical structure, are taken as the basis of this description.

The nodules of new-growth were very numerous, and many were confluent at their margins. They were circular in outline and ranged from a microscopical size up to $\frac{1}{8}$ inch in diameter.

The tumour-cells were for the most part arranged in single rows, or in double rows around a central lumen, an arrangement suggestive of a tubular carcinoma. This appearance was even more marked when the tubules were seen in transverse section.

Generally speaking, there was only a small amount of fibrous tissue between the rows of cells, and in places the cells rested directly upon the endothelium of capillary blood-vessels. At the margins of the nodules there was no capsule, the tumour-cells abutting directly upon liver-tissue.

The blood-vessels within the nodule were restricted to a few capillaries coursing between the rows of cells.

The tumour-cells showed considerable variations in size, and ranged in shape from cubical to a deep columnar. They had a delicate cell outline, and their protoplasmic contents stained faintly with acid dyes, and showed in varying degree the reticular structure characteristic of tumours of the supra-renal cortex, and due to fatty and perhaps also glycogenic deposition.

The nuclei were large and vesicular, the nuclear network was loose, and a central dot showing meta-chromatic staining was present. There were numerous mitotic figures in the cells.

The surrounding liver was compressed, the cells small and atrophic, and in places there was congestion of the intra-lobular capillaries.

In the left lobe of the liver scarcely any remnants of liver substance were present, the large white masses consisting of collections of tumour-cells separated by broad bands of dense fibrous tissue. The cells were less regularly arranged in rows and more fibrous tissue penetrated between them, whilst individual cells showed considerable degenerative changes, even to complete necrosis. Scarcely any blood-vessels were present.

The masses of growth on the peritoneum and the nodule in the neighbourhood of the pancreas showed the same features as the mass in the left lobe of the liver, and in places there was some formation of small cysts, with intra-cystic papillomatous growths.

The mass in relation to the pancreas was contiguous to, but did not invade, the parenchyma of the organ.

Remarks by Dr. Sidney Phillips.—The occasional existence of accessory supra-renal bodies in various situations—the kidney, the peritoneum, etc.—has been described by several observers.

The first examples of such aberrant "rests" in the liver-substance appear to have been those recorded by Schmorl (*Ziegler's Beiträge*, ix, 1891). In four bodies which he examined, in all of which the supra-renal glands were themselves quite normal, he found in the liver substance close to its surface two or three nodules of yellowish or orange tint consisting of supra-renal tissue.

Obendorfer in 1900 (*Centralb. für Allg. Path. und Path. Anat.*, Bd. 11, 1900) described two cases in which such aber-

rant nodules were found in livers affected by syphilitic lesions. Pepere (*Arch. de Medecine Experimentale et d'Anatomie Pathologique*, v. 14, 1902), in an article of great length and detail, summarises his conclusions on the subject as follows: that germs of adrenal capsules may, during embryonic life, become included in the substance of the liver tissue, conveyed there probably by the blood-vessels entering the organ; they may consist of cortical substance or of cortical and medullary substance, or more rarely of medullary substance only. The cells of these adrenal "rests" may give origin to neoplasms of epithelial type; the nodules thus arising reproduce the structure of adrenal tissue.

In the same article Pepere describes what appears to be the first recorded example of the development of malignant disease from such included supra-renal tissue in the liver.

My own case, recorded this evening, appears to be a similar case of primary carcinoma of the liver arising from an included "adrenal rest"; in addition to the growth in the liver there were many small nodules of similar growth in the peritoneum; but their small size and apparently very recent development compared with that of the liver growth renders it, I think, certain that the liver was the primary seat of the cancer.

In the naked-eye appearance of the liver there is nothing, I think, to distinguish it from a case of cancer of the liver such as ordinarily seen, but Pepere describes it as characteristic for the nodules to be of an orange-yellow colour.

The engravings given by Pepere show that the microscopic appearances of the growth in his case were exactly similar to those seen in the microscopic slides of my own case, shown this evening by Mr. Spilsbury, and of which a micro-photograph is appended to this paper.

In their clinical aspects the two cases were very similar. Both patients were females, æt. 40; in both the liver could be felt enormously enlarged and nodular; and in both there was very copious ascites and no jaundice (though in Pepere's case it is said there was a slight icteroid tint of skin); in neither case was there any pain, and both ran a very rapid course to a fatal issue.

It will be seen, therefore, there was little to distinguish these cases during life from other forms of carcinoma of the liver, though it is not common in carcinoma of liver for ascites to be present without jaundice. Pepere says they most resemble sarcoma of liver.

PLATE VIII.

Illustrating Dr. Sidney Phillips and Mr. B. H. Spilsbury's Case of Adrenal Carcinoma of the Liver.

FIG. 1.—Adrenal carcinoma in liver. Section of a small secondary deposit. × 80. Hæmatoxylin and eosin. *a*. Carcinoma cells showing imperfect tubular arrangement. *b*. Blood-vessel. *c*. Compressed and atrophied liver cells.

FIG. 2.—Adrenal carcinoma in liver. Section through margin of a secondary deposit. × 500. Hæmatoxylin and eosin. *a*. Carcinoma cells showing fine protoplasmic reticulum. *b*. Compressed and atrophied liver cells. *m*. Karyokinetic figure in carcinoma cell.

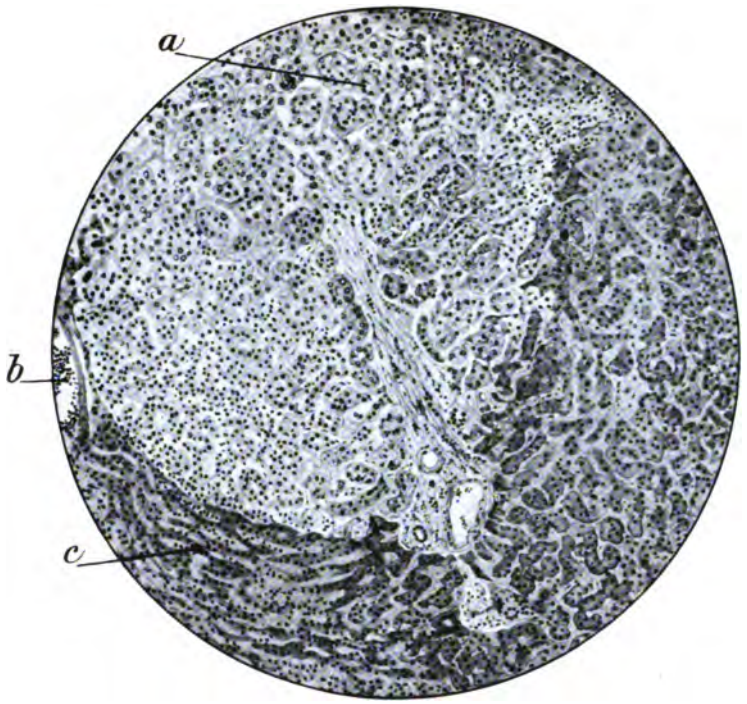

FIG. 1.

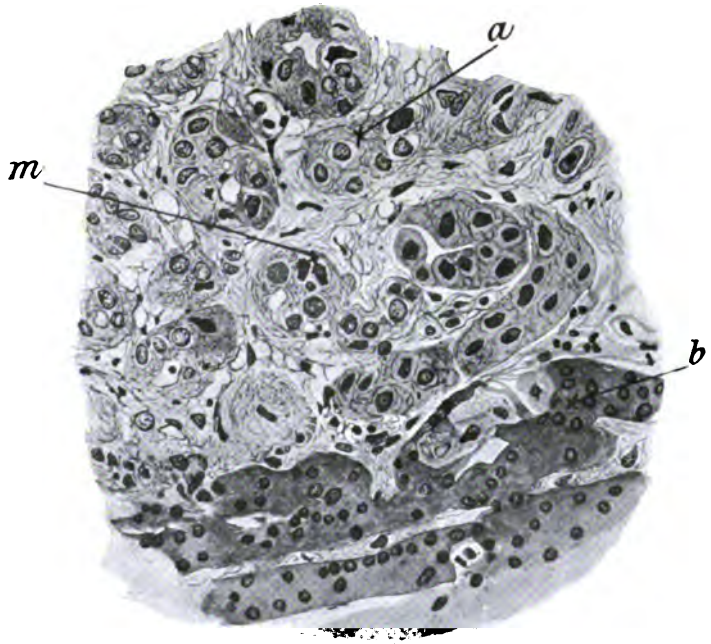

FIG. 2.

XXXII.—*A Case of Diabetes Mellitus in which Xanthoma supervened; Lipæmia; Fatty Necrosis of the Pancreas.* By Sir Dyce Duckworth, M.D., LL.D. *Read May* 26, 1905.

G. M., æt. 23, a clerk, was admitted into Matthew Ward, St. Bartholomew's Hospital, on December 6, 1904, suffering from diabetes mellitus. His first symptoms dated from September, 1904, and were thirst and wasting. He had previously considered himself strong. His parents were alive and well; he had four brothers and three sisters in good health, but had lost one sister from diabetes at the age of 31. No other cases of diabetes amongst his relations had occurred so far as known.

The patient was a dark-haired, sparely nourished man. His temperature was 97·4°, respirations 18, and pulse 72 per minute. The urine was of sp. gr. 1040, void of albumen, and contained 25 grains of sugar per ounce. Nothing abnormal was noticed in the thorax or the abdomen. The knee-jerks were present. The tibiæ were slightly curved owing to rickets in early life.

The diet was gradually made rigid in respect of exclusion of carbohydrate food. The appetite was very large, and about five pints of fluid were taken daily. The patient lost weight. About a fortnight after admission an eruption of a large number of papules occurred on both elbows, hard and shotty, painful on pressure, pink in colour. From day to day fresh papules appeared, spreading to the neck, back, and limbs, fairly symmetrical in character. Most of these grew larger, some becoming the size of a split pea, and manifesting a yellow-coloured summit.

By January 14, 1905, the sugar was slightly diminished in amount. The only medicines taken were a mixture of sodium bicarbonate with nux vomica and calumba, and an emulsion of cod-liver oil. An ointment of ammoniated mercury with solution of coal-tar was applied to the eruption. Exercise was taken daily in the hospital square until the patient became too feeble to avail himself of it. He steadily lost strength. The eruption showed no signs of involution. The iron reaction in the urine increased in intensity. Severe pains occurred in both deltoid muscles, and along the bicipital

grooves, but there was no evidence of neuritis. The arms became very feeble, and the patient slept badly. The papules became of a more yellow tint. In February there was a little gain of weight. Sodium bicarbonate was now given in full doses of ʒj, *4tis horis*, yet the iron reaction fully persisted in the urine.

On February 16 severe epigastric pain was complained of, followed by vomiting. Drowsiness set in, and severe dyspnœa. On the 17th there was coma, and the patient gradually sank and died on February 18. An examination of the body on the following day revealed the following conditions:

Post-mortem note on G. M., æt. 28.—Admitted December 6, 1904; died February 19, 1905. *Post mortem*, February 20, 1905.

Disease.—Diabetes mellitus: coma. Extensive fat necrosis of pancreas and surrounding structures. Xanthoma diabeticorum. Lipæmia.

External appearance.—On skin of both forearms, arms, backs of hands, buttocks, are small raised yellowish nodules of varying size, some as large as peas.

Head.—Dura mater and sinuses, cranial bones, arachnoid and pia mater, arteries: natural.

Brain.—50 oz.: natural. Spinal cord, eyes, ears: natural.

Chest.—Œsophagus, glands of neck, larynx and trachea: natural.

Lungs.—Right 27 oz.; left 25 oz.: natural. No signs of tubercle; at base, patches of fat necrosis.

Heart.—7 oz., dilated, valves natural.

Abdomen.—Intestines, mucous membrane of duodenum: natural.

Liver.—65 oz.; large, pale, rather friable: no fat necrosis is seen.

Spleen.—11 oz., large and soft. Abdominal lymphatics, suprarenals: natural.

Kidneys.—10 oz. together: natural.

Blood.—From jugular veins and right auricle, milky in appearance.

Chyle.—From thoracic duct, very abundant and milky, flowing freely on section.

On lifting the stomach, which was dilated (holding about Oiij) the peritoneum lying between the stomach and transverse colon and extending to the mesentery, was seen to be

covered by a putty-like substance which did not wash away, but was in the substance of the membrane.

The reflexions of the peritoneum over duodenum and right kidney, also the tissues at the hilum of the spleen, were in a similar condition.

The focus of this fat necrosis was the pancreas, from which the process seemed to have spread out in all directions.

The *pancreas* itself was about the usual size, but hard to the feel, and on section showed about half its substance converted into the material under the colon, of the colour and consistency of half-dried putty.

A coloured drawing of the eruption was made, and some of the papules were excised during life and examined by Dr. Andrewes, who reports as follows:

"The epidermis is thickened a little over the papules, the thickening being in the Malpighian layer and not in the stratum corneum. The chief changes are seen in the dermis, which shows large groups of rounded cells with a clear nucleus lying amongst the strands of fibrous tissue. These groups are in the vicinity of the sweat-glands and hair-follicles, but do not seem to have any specific connection with these structures. They are most strikingly brought out by osmic acid staining, which shows the rounded cells to be densely packed with small fat globules, the nucleus appearing as a clear area. They differ from ordinary fat-cells in containing many small fat globules instead of one large one; they show no sign of being degenerate.

"I do not feel able to state any confident opinion as to the origin of these fat-laden cells, which are clearly the specific element in the lesion. They are presumably either endothelial or of connective-tissue origin. In addition to these cells there are a few ordinary fat-cells in the corium and a certain amount of free fat, probably due to the breaking down of the specific cells. The sweat-glands and other accessory structures of the skin appear normal."

Dr. Luff kindly suggested to me, when I showed this patient to the Society on December 20, 1904, that the xanthoma was possibly dependent on the irritant effects of β-oxybutyric acid in the blood. The urine was therefore examined with a view to detect its presence, and the following report by Dr. Hurtley is subjoined:

"It gave strong ferric chloride reaction. It gave strong reaction for acetone. Examined for β-oxybutyric acid as follows: 1000 c.c. made just alkaline with sodium carbonate

and evaporated nearly to dryness on water-bath. Treated with sulphuric acid and liquid distilled in a current of steam, a very small amount of needle-shaped crystals collected in condenser.

"The whole distillate was extracted with ether, extract dried, ether distilled over. A yellowish residue-liquid remained. This was treated at 160° exactly, to expel fatty acids. A very minute residue remained, too small to take a melting point or prepare a derivative, but it gave acid reaction to litmus.

"The conclusion to be drawn is: (1) that a fair amount of fatty acids was present; (2) in all probability a very small trace of β-oxybutyric acid was present.

"The method employed (Külz's) was the best that could be used in the absence of a polarimeter, but I incline to think there is a loss of α-crotonic acid on heating at 160°."

The condition of lipæmia was not ascertained during life. It is probable that an inspection of the retinal arteries might have revealed it, as it did in Sir Thomas Fraser's patient whose case is so admirably reported in the *Scottish Medical and Surgical Journal*, September, 1903, and in others recorded by Drs. Starr, Glynn, and Fuchter. The excretion of sugar diminished during the last two days on the supervention of toxic symptoms, as has often been observed. The temperature remained subnormal throughout the patient's sojourn in the hospital.

Cases of Xanthoma diabeticorum are still sufficiently rare to be worthy of record. The conditions of lipæmia and fatty necrosis of the pancreas are seldom met with, although fatal cases of diabetes are frequently inspected in the dead-house. It is important to note that this variety of xanthoma may occur in persons who are not diabetic. It has been met with in cases of intermittent glycosuria, and is certainly more common in males than in females.* With respect to the ætiology of the disorder, it may fairly be believed that the irritation of the skin arises from the presence of some toxic element which is generated as a result of an altered hepatic or pancreatic metabolism.

* Vide *Diseases of the Skin*, edit. iii, vol. ii, p. 695; Dr. Radcliffe-Crocker with reference to other cases. The first recorded instance was that of Dr. Hughes, which occurred in Guy's Hospital in 1848, and a model illustrating the eruption is preserved in the Museum of that Hospital.

PLATE IX.

Illustrating Sir Dyce Duckworth's paper on Xanthoma Diabeticorum.

Arm of patient, showing the appearance of the papules of Xanthoma Diabeticorum about one month after their formation.

Bale & Danielsson, Ltd lith.

XXXIII.—*Cases of Stokes-Adams' Disease.* By A. M. Gossage, M.B., F.R.C.P. *Read May* 26, 1905.

WHAT has been called Stokes-Adams' disease is in reality merely a collection of symptoms due to a variety of pathological causes. The clinical aspect of these cases is, however, so distinctive, and the prognosis is so grave that the condition deserves a special name, and a more suitable name than that given by Huchard (1) has not yet been proposed. The condition is certainly uncommon, but not extraordinarily rare; at any rate, about 100 cases have been recorded in medical literature on the Continent and in America. The distinctive features are (*a*) an extremely infrequent pulse, which is usually conjoined with a similar infrequency of the heart's action, although in some cases at certain periods intermediate beats of the heart too weak to be felt at the wrist may be heard; and (*b*) certain nervous symptoms. The nervous phenomena may be divided into three groups: (*a*) Syncopic attacks in which the patient falls down suddenly, with complete loss of consciousness, feeble pulse and shallow breathing, to recover after a few minutes; these severe attacks are often preceded by, or may alternate with, slighter attacks of vertigo; (*b*) epileptiform seizures in which, in addition to sudden unconsciousness, there is some twitching of the muscles of the limbs and face, or even a generalised convulsion; (*c*) apoplectiform seizures, in which the sufferer falls suddenly into deep coma, with stertorous breathing and cyanosis, these attacks not being followed by any motor paralysis. The cases recorded here are types of the first two varieties of these nervous seizures, whilst the apoplectic form is fully exemplified in the original papers of both Adams (2) and Stokes (3) and also in Osler's (4) recent contribution.

The cause of these remarkable nervous attacks and of the infrequency of the pulse is very obscure. The majority of cases occur in males during the degenerative period of life in association with considerable sclerosis of the arteries. Stokes (3) considered the phenomena due to fatty degeneration of the heart, and it is probable that the cases met with after acute specific fevers are primarily caused by acute degeneration of the cardiac muscle (4). Huchard (1) describes the condition as "the cardio-bulbar form of arteriosclerosis," and considers the symptoms to be caused by a sclerosis of the arteries of the spinal bulb. Such an explanation, however,

would exclude those cases occurring in younger persons, in some of which valvular lesions have been found in the heart, in others gross lesions of the nervous system (5), while in the remainder no disease of either the vascular or nervous apparatus has been found after death (6).

As a rule, the infrequency of the pulse is permanent, but certain patients who must be included in the same class have an infrequent pulse only during their nervous attacks (1). Amongst the majority the pulse is generally much less frequent in the attacks than at any other times; indeed, Osler records a case where during an epileptiform attack no sound could be heard over the heart and no pulse felt at the wrist for 35 seconds. The ordinary causes for acceleration of the pulse—exertion, emotion, etc.—seem to affect these patients but little; sometimes, indeed, they cause a diminution in the rate. Drugs, such as digitalis and atropine, are said not to influence the pulse. Huchard (1) relates that an attack of pneumonia in one patient only raised the pulse rate from 36 to 44, although the temperature rose to 40° C. Nearly all the sufferers complain of feeling the cold and of difficulty in getting their hands and feet warm.

The prognosis is very grave, most of the cases tending to die suddenly in one of the nervous attacks. Cheyne-Stokes breathing, cardiac asthma, and angina pectoris occur, as might be expected, in some of the patients with arterio-sclerosis, while others may die of renal disease. In rare instances the duration is very prolonged; Osler (4) records the case of a man who had a permanently infrequent pulse and fainting attacks for 30 years and then died at the ripe age of 74 in one of his fainting attacks. The cases that occur after acute fevers not uncommonly end in recovery of health, and even cases with arterio-sclerosis may lose their infrequent pulse and nervous symptoms and live on in comfort (4).

Case 1. *Permanently infrequent pulse, with attacks of sudden unconsciousness, associated with muscular twitching.*—The following case was exhibited at the meeting of the Clinical Society on April 28, 1905:

S. C., a married woman, æt. 58, came to the Westminster Hospital at the end of March, 1905, complaining that for the last month she had suffered from attacks of "giddiness" three to four times a week. The patient was decidedly dull in intellect and was unable to give a clear account of her symptoms, but later a description of the attacks was obtained

from an eye-witness of them, the patient's daughter. The seizures came on suddenly, without warning, causing the patient to fall down in a condition of apparent unconsciousness; "she then becomes a sort of dead colour and goes all of a twitch," as the daughter expressed it. On recovering the patient was wont to declare that although she had been unable to speak she had yet been quite conscious of what was going on round her. It is probable that in this supposition she was mistaken, and that she was unconscious for at least a few moments. The attacks usually lasted between five and ten minutes. Besides these nervous attacks, the patient complained of feeling the cold and that her hands and feet frequently "went dead." About twenty years before a "piece of bone" had come from her nose.

On examination she had an apathetic appearance, and the bridge of her nose had sunk down owing to a syphilitic necrosis of the septum. The diagnosis of old syphilitic disease was confirmed by the presence of some scarring about the fauces. Nothing abnormal was found on physical examination of the lungs and abdomen. The radial arteries were rather thickened and the pulse fairly strong. On counting the pulse it was found to be beating at the rate of 40 to the minute, a beat being dropped occasionally. The heart was normal in size and the sounds were quite normal, except that the first sound was rather booming. The heart also was beating at the rate of 40 per minute, and no intermediate beats, too weak to reach the wrist, were heard.

On April 25 she was admitted into the hospital, and during her three weeks' stay the pulse-rate varied between 30 and 36, with an occasional rise to 40 or 44. On every occasion the heart and pulse exactly corresponded. On first admission a small quantity of albumen was found in her urine, but none was present on subsequent examinations. The patient expressed herself as feeling much better for her sojourn in the hospital; she certainly looked much healthier, seemed more intelligent, and was more alert in manner. As is frequent in cases of this kind, the nervous attacks were conspicuous by their absence during this period of rest and quiet. (7) She once had a transient attack of vertigo without loss of consciousness, in association with which her pulse-rate increased to 40.

Case 2. *Permanently infrequent pulse, with syncopic attacks; death in an attack.*—G. C., a clerk, æt. 23, was

sent to me in 1899 by Mr. Muirhead Little. He complained that for some months he had had occasional attacks of fainting, with unconsciousness, that his hands and feet were constantly cold, and further, that he was conscious of the beating of his heart at the rate of about 32 per minute. About five years before he had fallen on to the top of his head, and since this accident he had been unable to properly raise his eyelids.

The patient was very small, about 5 ft. 2 in. high, and slightly built, and there was some lateral curvature of the spine. There was partial ptosis of both eyelids. The apex beat of the heart was in the sixth intercostal space, an inch external to the nipple, and the beat was heaving in character; over the second right costal cartilage there was a systolic bruit, but no thrill. The pulse was of fair volume, normal tension, regular, 36 to the minute; the vessel walls were elastic, and showed no signs of sclerosis or thickening.

For three weeks he was an in-patient at the Westminster Hospital, under the care of Dr. de Havilland Hall, who has kindly permitted me to make use of his notes. According to these notes the pulse-rate during this time was always between 30 and 36 per minute, except on one occasion, when it fell to 28. The cardiac murmur varied a good deal, being sometimes audible and sometimes inaudible. He had no fainting attacks while in the hospital.

During the next three years he came to see me at intervals of three to four months. He had given up work and lived quietly at home, with the result that his general health had improved. He still, however, had occasional attacks of faintness, and according to his own account his pulse was always between 30 and 36 per minute; this was certainly the case whenever I counted it. He declared that he did not now lose consciousness in the attacks of faintness, but he frequently fell and sometimes injured himself. In March, 1901, when on his way to see me, he had an attack while getting off an omnibus; he fell, with the result that I had to dress a severe scalp wound. The rhythm of the heart and pulse always corresponded, and no slight intermediate beats of the heart, uncommunicated to the wrist, were ever noticed. On one occasion, in June, 1901, there was intermission of the pulse and heart every fifth beat, the rate being 29 per minute. On this occasion a murmur was heard with the systole over all the valves, being loudest over the pulmonary.

This patient died early in 1903 in one of his fainting seizures.

REFERENCES.

(1) Huchard: *Maladies du Cœur et des Vaisseaux,* 1893, p. 309.
(2) Adams: *Dublin Hospital Reports,* iv, 1827, p. 396.
(3) Stokes: *Dublin Quarterly Journal of Medical Science,* 1846.
(4) Osler: *Lancet,* 1903, August 22.
(5) Holberton: *Medico-Chirurgical Transactions,* 1841.
(6) Edes: *Transactions of Association of American Physicians,* 1901.
(7) Jaquet: *Deut. Archiv. f. Klin. Med.,* lxxii.
Jaquet gives a fairly full bibliography.

Addendum.—The following case came under the author's care at a later date:

CASE 3. *Infrequent pulse, with frequent attacks of vertigo and occasional syncopic seizures and unconsciousness.* — E. C—, a shoemaker, æt. 69, came under my care at the Westminster Hospital on June 15, 1905. He had been perfectly well until March 1, when he was suddenly attacked with unconsciousness while in the street; he fell to the ground, and was conveyed to the Casualty Department of the Hospital, where he rapidly recovered and was soon allowed to go home. Since this first seizure he had had frequent attacks—several a day—of giddiness. Some of these were merely momentary, but others were more severe, causing him to fall backwards, and sometimes to injure himself. He had one of the slighter attacks in my presence, but, except for his own statement, I should not have been aware of his feeling giddy, as there was no alteration in his appearance nor in his pulse. The patient's son informed me that on one occasion, in the middle of May, he was with his father when the latter suddenly turned to him and said that he felt queer; "my father then fell down unconscious, his face became very pale, but there was no twitching of his limbs; he remained unconscious for five minutes." The patient described himself as a very excitable man, and confessed to taking large quantities of snuff.

On examination he was found to be decidedly anæmic, the mucous membranes being very pale. The heart was overlapped to some extent by lung, and the heart-sounds were

distant. No enlargement of the heart could be made out. The vessels were markedly atheromatous and the pulse tension was a little above the normal. The heart and pulse were synchronous and were both beating at the rate of 32 per minute. The urine was acid, 1012, and contained a fair quantity of albumen.

On June 24 he had an attack of unconsciousness, lasting ten minutes, in which there was distinct twitching of the muscles of the face and upper limbs. On June 29 the pulse rate was 84.

CLINICAL CASES

EXHIBITED.

*** Published in accordance with the regulation relating to the exhibition of patients at the meetings of the Society, viz. that each case shall be accompanied by a brief written description of the points it illustrates; such description to be published or not in the *Transactions* as the Council may direct.

I.—*Case of Acute Ataxia (Encephalitis cerebelli).* By Frederick E. Batten, M.D. *Exhibited October* 28, 1904.

J. E., æt. 3½. Had measles in March, 1904, up to which time he had been perfectly well. On April 2 he was quite bright, and got up and sat in a chair. He gradually got dazed, vomited, and at 8 p.m. was unconscious and remained so till April 7. He had several general convulsions. On April 9 he was quite conscious, but his arms and legs shook and his left eye moved continually. He could not sit up and could not speak.

Family history.—Father has phthisis. One child has died of tuberculosis. The boy was first seen on May 13. He was well nourished, and appeared intelligent. He spoke in a slow, hesitating manner. He was unable to sit up in bed without support, and when put on his legs walked in the most incoordinate manner. There was marked inco-ordination of both hands, but the grasp was fairly good and equal on the two sides. There was no facial weakness, no ocular paresis or nystagmus, and the optic discs were normal. The knee-jerks were active. There was a tendency to ankle clonus on both

sides, and the plantars generally gave flexor responses; on one or two occasions a distinct extensor response was obtained. The boy swallowed well, and there was never any regurgitation of fluid. During the three months he was under treatment only slight improvement took place, and when discharged he was still very ataxic.

After returning home the boy complained of pins and needles in the feet and hands, no cause for which could be found.

The mother states that since his illness the boy has become most untruthful. Not only does he tell lies to obtain his purpose, but he invents stories which can obtain him no possible advantage. His moral sense is perverted.

II.—*Case of Acute Ascending Paralysis, followed by much Improvement, with Microscopic Demonstration of the Tetracoccus found in the Spinal Fluid.* By E. D. MACNAMARA, M.B. *Exhibited October* 28, 1904.

W. H., æt. 30, a waiter. Admitted to the Westminster Hospital (under the care of Dr. Murrell) on August 3, 1904, with a history of rapidly-increasing loss of power in the legs, which had begun eleven days previously and had culminated in absolute palsy in three days' time. For three days before admission the patient had noticed gradually increasing feebleness of the arms, difficulty in swallowing, and double vision.

There is a history of a thoroughly treated attack of syphilis at 24.

On admission there was a total flaccid palsy of both legs, except of the right hallux, inability to rise from the bed, much loss of power of the arms, defective articulation, defective ocular movements, and both pupils were almost immobile to light. No reflexes were obtained from the lower limbs: otherwise superficial, deep, and organic reflexes were normal. There were no sensory subjective symptoms or objective signs. The electrical reactions were normal.

On August 8th slight signs of improvement were noticed, and patient has since made steady progress to recovery. Articu-

latory and ocular symptoms have passed off, and the power of the arms has become good. Patient cannot yet walk, but can move his legs freely; his pupils react well to light, and his knee-jerks are returning.

III.—*Two Cases of the Peroneal Type of Family Amyotrophy (Tooth's Neuritic Muscular Atrophy).* By J. H. Bryant, M.D. *Exhibited October* 28, 1904.

CASE 1.—Lilian S., æt. 12, was first admitted into Guy's Hospital under my care for wasting of the legs and hands, on April 11, 1901. Forceps had to be used at her birth, but it did not appear that the brain was injured, for at the age of nine months she could walk. Although a small child, she was considered to be strong and healthy. At the age of 10 months she had bronchitis, and during the next three years had several other attacks. When 4 she suffered from whooping-cough, being ill for three weeks, at the end of which time she was unable to walk. On account of this trouble she was taken to an orthopædic hospital, where a diagnosis of infantile paralysis was made, and she was ordered to wear boots with iron supports. At this time both feet and legs were wasted up to the knee, the right more so than the left, and on attempting to walk her knees would give way without any warning.

When 5½ she had scarlet fever, and at 6 and 8 attacks of measles. In January, 1900, her mother noticed for the first time that her hands and body appeared to be getting thinner. At this time she also complained of aching pain in her feet, and could not walk as far as she could a month or two before. The wasting of the muscles of the hands had gradually progressed, and she found she could neither hold nor pick up small objects as well as other children. She had never had a fit of any kind, and has always had perfect control over her bladder and rectum.

Both her parents appear to be strong and healthy.

The father stated that one of his brothers suffers from weakness and wasting of the muscles of his hands, arms, and legs; that he has a sister who suffers from epileptic fits, and that another of his sisters is affected with heart disease.

Both his mother and father are well, and with the exception of his brother there is no history of muscular atrophy in any other member of the family. His wife has had nine children since their marriage, of whom five are living. They were strong and healthy until three years ago, when one of the boys, æt. 8, who is the fourth living child, was noticed to be weak in his legs. Two sons, æt. 18 and 17, are quite well.

The history of the uncle is as follows: He was late in learning to walk. When three years of age weakness commenced in his feet, without any apparent cause. Wasting of the muscles followed, and at the age of 10 his hands became affected in a similar manner with weakness and wasting. From this time the trouble has been progressive in character. He is now æt. 42 and is almost helpless, the wasting having extended from the feet to the knees, and from the hands to the forearms. At first he was able to get about with the help of a stick, then he had to resort to crutches. Three years ago his condition was described as follows: "He is quite helpless as to his hands and feet, and wears pads on his knees, dragging his feet and legs behind him as he moves along, whilst his hands are so weak that he is quite unable to hold his short crutches, to remedy which defects, iron hooks have been attached to his forearms, and with these he grasps the crutches. He appears to be strong and healthy in other respects, without wasting of his body, upper arms, or thighs. At the present time his only method of progression is by crawling on his knees and hands.

Condition of the patient in April, 1901.—When lying in bed she appears a healthy, intelligent, and bright-looking child. If the arms are raised, a condition of wrist-drop at once becomes apparent. There is marked wasting of the thenar, hypothenar, and interossei muscles of both hands. The forearms are slightly wasted. The muscles of the trunk are not atrophied.

The scapulæ are rather prominent, and there is slight spinal curvature in the dorsal region with the convexity backwards, and slight lordosis in the lumbar region. She has the full use of her arms, free movements of her hands, and a very fair grip, considering the amount of muscular atrophy.

Neither the deltoids, scapular muscles, nor facial muscles are wasted.

As she lies in bed the feet are extended, there is slight talipes varus and marked wasting of the muscles below the knees. The thigh and gluteal muscles have not atrophied.

None of the muscles of the body are large or hard, and there is no history of any of the muscles having been previously enlarged. The gait is of a high-stepping character. There is slight genu valgum. The left leg is weaker than the right, but she can raise both legs from the bed against a fair amount of resistance. The movements of the knee and hip joints are good.

The movements of the thumb are rather weak. There does not appear to be any weakness of the upper arm muscles or of the shoulder or scapular muscles. There is no spasm, tremor, or inco-ordination. Slight fibrillation has been noticed in some of the muscles. There is no loss of sensation to touch, pain, heat, or cold, and all of these forms of sensation can be appreciated and differentiated.

The knee-jerks cannot be obtained. There is no ankle clonus. Wrist and elbow jerks cannot be elicited. The plantar reflex is present and is flexor in character. The abdominal, epigastric, and scapular reflexes are present.

With the Faradic current there is no response in the thenar and hypothenar muscles and the interossei, with the exception of the third left dorsal, in which a slight contraction can be obtained. The flexor and extensor muscles of the forearm respond normally, also the scapular, shoulder, upper-arm, and facial muscles.

The small muscles of the feet, the peronei, tibialis anticus, and extensor longus hallucis do not react to Faradism, but the calf muscles, the glutei, and thigh muscles react normally. Those muscles of the leg which do not react to Faradism do not react to galvanism, but on stimulating other muscles K.C.C. is greater than A.C.C.

There are no changes in the optic disc, retina, or choroid. The pupils react to light and accommodation.

The circulatory, respiratory, and abdominal systems do not present any abnormal physical signs.

Present condition.—With the exception of more wasting of the thenar, hypothenar, and interossei muscles, the disease does not appear to have progressed much since April, 1901. It is surprising to find she can walk as well, if not better than she could three years ago. This improvement in walking appears to have been noticed after her recovery from an attack of scarlet fever which she contracted at the end of last year.

There is more deformity of the right hand, which now presents a well-marked "claw-like" appearance, which is

owing to the proximal phalanges being kept extended, while the middle and distal phalanges are permanently flexed. The thumb is more extended. The left hand is in a somewhat similar but less marked condition, the flexion of the fingers being much less pronounced.

The legs are thin and wasted and have a broomstick appearance. There is well-marked foot-drop. There is no voluntary movement at the ankle-joint, but the foot can be dorsiflexed, showing there are neither contractions nor adhesions.

On testing the electrical reactions of the muscles of the hands one striking fact was noticeable, viz. the apparent loss of sensibility to electrical stimuli—for, even with a very strong current, the child complained of neither pain nor discomfort.

The muscles of the hands reacted to neither Faradism nor galvanism.

Case 2.—Percy S., æt. 8, was first admitted into Guy's Hospital in August, 1903.

At his birth forceps were used and a good deal of difficulty was experienced. He was delicate up to the age of 12 months, when he suffered from diarrhœa and vomiting. When 3½ he suffered from "English cholera." He was able to walk and run about as other children, however, until about two and a half years ago, when, after an attack of measles, his mother noticed that he was weak in his legs and that he could not lift his big toes. One foot was worse than the other when he was first affected, but the mother does not remember which it was. He seemed to be helpless and began to fall down frequently on account of his legs giving way under him. Wasting of the muscles of the legs was next noticed, and he began to complain of pain in his back, at the top of his head and in the abdomen.

When first seen, both legs were decidedly weak, and he could not flex his knees against very slight resistance. The most striking feature of his condition was the dropping of his big toes. He had lost the complete power of extending both his big toes, and it appeared as if the extensor longus hallucis of each leg were the first muscles to be affected. The calf muscles were soft and flabby. The muscles of the hands were not affected.

Present condition.—He is much worse than he was a year ago. He cannot walk at all by himself unless his legs are supported by straight internal splints extending from the foot to just below the knee. The splints and the method of

applying them to his legs were devised by his uncle, who is suffering, most probably, from an advanced condition of the same disease.

The muscles of both legs are wasted, especially the extensors. The feet are dropped and the big toes are flexed. The feet are in a condition of talipes equino-varus, and there is well-marked pes cavus. He has no voluntary movement of the ankles.

The thenar and hypothenar eminences have become slightly more flattened, and it is evident that the muscles of the thumb and little finger are beginning to atrophy.

An attempt was made to test the electrical reactions of the muscles, but the child was so frightened and struggled and made such a noise that it was impossible to obtain any reliable data.

The pupils react to light and accommodation.

IV.—*A Case of Fibroma of the Left Scapula, Ribs, and Spine.* By J. Jackson Clarke, M.B. *Exhibited October* 28, 1904.

AMY P., æt. 24. Patient had been treated by a "poro-plastic" jacket for nine months when she first came to the City Orthopædic Hospital, in October, 1901, complaining of pain in the left shoulder. On examination, the shoulder was found to be raised and completely fixed: there was no curvature of the spine. Between the vertebral border of the scapula, near the root of its spinous process and the spine was a firm, round lump (photograph shown). On cutting down to the lump under local anæsthesia (eucaine), I found a hard, white growth which, on histological examination, proved to be a dense fibroma. The patient was offered and accepted an opportunity of a tentative operation, with a view of, if possible, removing the growth, together with the scapula. An exploration was made through an incision skirting the vertebral border of the scapula. The growth was found to fill the ventral surface of the scapula, to which it was closely adherent, and to extend to the ribs, of which those on which the scapula rested were found to be surrounded by the growth, and both my colleague, Mr. Mayo Collier, and myself

decided that only a partial removal could be effected. This is now three years ago. At the present time the growth extends along the vertebræ to the lower cervical region, and the patient suffers considerably from pain caused by pressure on the nerve-roots.

V.—*Cases of Sprengel's Shoulder.* By H. A. T. Fairbank. *Exhibited October* 28, 1904.

Case 1.—Winifred W., æt. 4. No history of any other deformity in family. The child appears to be normal in every way with the exception of the right shoulder, which was noticed ten months ago.

Right shoulder.—The right scapula is situated at a higher level than the left. It is also nearer the mid-line when the arms are hanging by the side. The angle is rounded; the distance from the spine of the scapula to the angle is distinctly less on the right side than on the left. The mobility of the right scapula is diminished: when the arms are held horizontally forwards the right scapula does not move outwards and forwards like the left does. The right arm cannot be raised quite to the vertical position; this is due apparently to some fixation of the scapula, and not to any defect in the shoulder-joint.

There is a band (? fibrous) passing down from the superior curved line of the occipital, one inch to right of mid-line, to the upper part of the vertebral border of the scapula. The left scapula is normal.

There is some rotation of dorsal vertebræ to the right.

No paralysis can be made out of any muscles.

There is no asymmetry of the face, and no torticollis.

Electrical reactions.—Serratus magnus and trapezius respond less readily to Faradic currents on the right than on the left side, and these muscles on the right give a sluggish contraction with both Faradic and galvanic currents.

X rays.—First and second ribs appear to be fused on the right side. The third is also slightly irregular in shape.

Case 2.—Alice S., æt. 12. She is the second of four children; one died of measles, the other two are healthy.

PLATE X.

Illustrating Mr. Fairbank's cases of Sprengel's Shoulder.

Skiagram of thorax of case of Sprengel's shoulder (girl, æt. 4), showing first and second ribs on right side fused, and others slightly deformed.

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PLATE XI.

Illustrating Mr. Fairbank's cases of Sprengel's Shoulder.

Skiagram of thorax of case of Sprengel's shoulder (girl, æt. 12), showing deformity of the spine, and the irregular arrangement and imperfect ossification of the ribs.

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The mother had five healthy children by a previous marriage. There is no instance of any deformity in the family. The left shoulder was noticed to be raised at the age of 3 years. No history relating to the paralysis of right leg can be obtained. *Left scapula* is situated at a higher level than the right. It is much nearer the spine than the right, when the arms are by the side. The angle is rounded; the distance from the spine of the scapula to the angle is less on the left than on the right side. The mobility of the scapula is greatly diminished, and this is seen in all movements of the arm, although the shoulder-joint itself is normal. No definite fibrous band or bony ridge can be found to account for this fixation of the scapula. The infra-spinatus is somewhat wasted. The *right scapula* is normal in size and shape; it is slightly winged.

The lower fibres of both trapezii appear to be weak. The right serratus magnus is weak. There is distinct deviation of the vertebral spines from the mid-line, the convexity being to the left in the dorsal region. A compensatory curve to the right is present in the lumbar region.

The left ribs are deformed, being more prominent anteriorly than the right, *i.e.* the exact opposite of what would be expected with such a spinal deformity. The mobility of these ribs is impaired. Both arms are normal.

The right leg is wasted and cold; this is due apparently to infantile paralysis. The right knee-jerk is absent. The left knee-jerk is brisk.

There is slight asymmetry of the face. The chin deviates slightly to the left. The right side of the face is slightly flatter than the left.

Electrical reactions.—The left serratus magnus and trapezius muscles respond less readily to galvanic and Faradic currents than do the same muscles on the right. There is *no* reaction of degeneration.

X rays.—Show great irregularity of the upper ribs on both sides. The ossification of the ribs is very imperfect on the left. On the right side the third, fourth, and fifth ribs are fused a short distance from the spine.

VI.—*A Case of Congenital Dislocation of the Right Hip-joint.* By J. Jackson Clarke, M.B. *Exhibited October* 28, 1904.

THE patient, Amy T., æt. 5, came to the City Orthopædic Hospital on June 23, 1903. She had marked shortening of the right lower extremity, lordosis, and lameness which was noticed as soon as she began to walk. A delicate child with nasal obstruction, chronic fever of 1°, poor appetite. The dislocation was reduced by Lorenz's manipulative operation on August 20, 1903, in the out-patient room. The limb was put up in plaster of Paris for six months, the patient being allowed to walk about with a high (1½-inch) boot on the left side. When the plaster case was removed, the limb retained the position shown in the Skiagraph. The tissues, ilio-tibial band, etc., passing from the anterior superior spine to the thigh had contracted, and they prevented any diminution of the abducted position. In order to secure the patient as far as possible, matters were left thus, patient being allowed to walk as before, the mother being directed to rub the limb. On September 30, 1904, the band of contracted tissues was divided subcutaneously, and the limb brought to its present position, *i.e.*, abduction about 30° (Skiagraph shown). Both skiagrams show that the reduction is complete. The child walks with the limb half everted and held stiffly. Whereas formerly she dipped down when the right limb took the weight of the body, now she rises on that limb; the abducted position and the restored 1½ inches of length producing this result. The stiffness was found to disappear entirely when patient was anæsthetised for the purpose of dividing the band. It may be attributed to the tension of the muscles (ham-strings, etc.) that were previously shortened and to reflex influences arising in the previously disused acetabulum. A few adhesions may be present in some cases owing to rupture of the ligamentum teres. The stiffness is more marked in a nervous child than in those of more robust type. Some passive rotation can be obtained when the child is not alarmed.

In another case, operated on on January 14, 1903, this same stiffness was present for a time, but had, together with most of the abductions and eversions, passed off within a year of the operation.

The writer would divide the course of events after a successful manipulative operation into four stages—

(1) Six months' retention in plaster or other apparatus.

(2) Six months', or more, massage without any forcible attempts to diminish abduction and eversion.

(3) Diminution of abduction and eversion by gentle rhythmical passive exercises, aided, if necessary, by subcutaneous sections of the contracted band below the anterior superior spine, and manipulation of the joint (to rupture any adhesions that may be present). This period also lasts about six months.

(4) The remaining period is left to Nature. The use of the limb develops all the functions of the normal joint.

During the whole period the child is allowed and encouraged to walk, save for a few days at the beginning of Stage 3 if the small operation there mentioned is required. The third stage should end between eighteen months and a year from the time of operation.

The exercises and rubbings required are of quite simple character.

VII.—*Single Congenital Dislocation of Hips, Left Leg.* By E. Noble Smith. *Exhibited October* 28, 1904.

M. F., girl now æt. 6¾, operated on by Paci-Lorenz method, January 8, 1904, *i. e.* nearly ten months ago, when 6 years of age. Adductors divided by subcutaneous section. Limb retained in abducted position in special splint three months. The time of fixation was not long enough, and there was slight relapse, so a refixation was necessary.

VIII.—*Double Congenital Dislocation of Hips.* By E. Noble Smith. *Exhibited October* 28, 1904.

E. J., girl, now æt. 7½, operated on by Paci-Lorenz method January 20, 1903, *i. e.* eighteen months ago, when 6 years of age. Adductors divided by manipulation. Limbs retained in abducted position for six months. Subsequent progress good, but retarded by severe double pneumonia. Joints now firm in normal position. Walks fairly well, but with feet turned out. Is improving rapidly.

IX.—*A Case of Ulcer of the Tongue, and Destruction of the Palate of Uncertain Origin.* By A. H. TUBBY, M.S. *Exhibited October* 28, 1904.

ARTHUR B., æt. 22, a carman by trade, was first seen in the Out-Patient Department of the Westminster Hospital on May 30, 1904.

The affection commenced nine months previously with deafness and a sense of fulness in both ears. Then the nose became obstructed, and a discharge came from both nostrils. Speech became indistinct about a month or six weeks after the onset of the trouble.

No history or signs of syphilis, either congenital or acquired. The father and mother are in fair health, but there is consumption on both sides of the family. The patient is said to have had bronchitis when a child, but no other serious illness. Two sisters have died, but the patient does not know the cause of death.

The patient has been under observation in the Out-Patient Department since May under the care of my colleague, Mr. Arthur Evans. He has been treated with mercury and iodides, and had local applications of chromic acid.

Present condition.—He is a somewhat weakly-looking young man, and not well nourished. The speech is nasal, and articulation is imperfect, especially as to the letters b, p, r, and k. On examining the oral cavity it is at once seen that the soft palate has been destroyed, and that the under surface of the mesial and posterior portion of the hard palate is tuberculated and is constantly covered with tenacious muco-pus. The tonsils are ulcerated, and the posterior pharyngeal wall has been the seat of extensive shallow ulceration, and now presents an irregular surface, to which crusts adhere. The first appearance of disease in the mouth was as a mass in the tongue, and this has become an extensive irregular ulcer, with a raised broad edge, firm, but not hard, and sharply defined from the soft surrounding tissue. It is slightly tender, and has spread rapidly. Both nostrils are clear, and on examining with the laryngoscope the glosso-epiglottic folds are not invaded, while the epiglottis and larynx are normal, but anæmic. The cervical glands on both sides are enlarged, but not so greatly as when first seen.

(For the above notes I am indebted to our Surgical Registrar, Mr. E. Rock Carling.)

Dr. Hebb kindly examined both the discharge from the ulcer and portions of its edges. No tubercle bacilli were found in the discharge. Sections cut through the mucosa and submucosa gave evidence of inflammation of some intensity. The surface is in a condition of ulceration, and the subjacent tissue shows inflammatory exudation and œdema. Except on the surface, where fungi and bacteria are present, no micro-organisms were detected.

Special examinations were made for bacteria, including tubercle bacilli, actinomyces, and blastomyces.

The following diagnoses have been suggested: Syphilis; but the absence of all response to treatment for this disease negatives it; nor does it appear to be actinomyces. Chronic glanders, as the man is a carman, might be entertained, but the history and appearances seem to consort more with lupus or tubercle.

Since this was written the ulceration has made most rapid progress, and the gums of the upper jaw are now invaded.

Additional notes.—The patient was readmitted on January 22, 1905. The tongue, mouth and pharynx were in much the same condition as when he left hospital nine days ago. On January 14 an injection of mallein (mxvi) was given at 2.20 p.m. This was followed by an abrupt rise in the temperature from 98·4° to 100·8° at 7 p.m. The temperature fell by degrees and reached normal by 7 a.m. on the 16th (in 36 hours), and remained normal.

On the 17th a second injection was given at 2 p.m., and the temperature again rose to 103·4°, and fell to 97·6° by 7 a.m. on the following day.

On January 21 there was an evening rise of temperature to 100°, on the 22nd to 99°, on the 23rd to 100°, on the 24th to 99·6°, and on the 25th to 101°.

On January 26 an injection of tuberculin was given, and the temperature rose to 100·2°, only there was no local reaction. For a time the tongue seemed benefited by leaving it alone, but lately the disease has made rapid progress. From January 28 to January 30 the temperature has been irregular, sometimes 100·4°, at other times 101·4°. On February 1 the temperature was normal. Dr. Hebb's examinations of the tissue removed gave no definite results, unfortunately.

It would seem from the above-mentioned effects of the in-

jections that the case may prove to be one of chronic glanders, but a further report will be made to the Society later.

The patient was re-admitted April 4, 1905. The pharynx, mouth, and tongue were *in statu quo ante*. Calomel injection gr. v was given thrice daily. Mouth-wash of listerine was given.

Present condition.—No soft palate present; the hard palate is covered with granulations. The alveolar processes of superior maxilla appear to be eaten away. The whole tip of the tongue is gone.

A scraping of the tongue was sent to the Lister Institute for the purpose of injecting guinea-pigs, and the following report returned: "Three guinea-pigs were inoculated on April 6 with an emulsion of the material received, as follows: (A) 2 c.c. subcutaneously; (B) 1 c.c. subcutaneously; (C) 0·5 c.c. intra-peritoneally. Pigs B and C appear to be well. Pig A died on April 18 (twelve days after inoculation) with very typical *post-mortem* signs of glanders (caseating tubules and nodules in peritoneum, etc.). Cultures made from these lesions showed bacilli which morphologically resemble glanders bacillus.

April 28.—Another of the pigs inoculated on April 6 died with three caseating nodules in abdominal wall, from which pure cultures of glanders have been obtained. These inoculations and their results clear up the case. It is one of chronic glanders. In the original communication it was surmised that among the four possibilities this was one. Unfortunately nothing more can be done for the patient, although it sometimes happens that cases of chronic glanders recover spontaneously, whereas acute glanders in man is always fatal.

X.—*A Case two years after Excision of the Sac of a Spina Bifida (Meningo-myelocele).* By W. G. SPENCER, M.S. *Exhibited October* 28, 1904.

A BOY, now æt. 2½, was operated upon when six months old. The cauda equina and nerves were dissected out and laid in the groove, which was closed by two rows of sutures. A firm scar now covers the groove, the legs are used well, and there is no hydrocephalus.

XI.—*A Case of von Recklinghausen's Disease (Multiple Molluscum Fibrosum with Pigmentation. Large Fibroma of the Hard Palate. Elephantiasis Mollis of Calf of Leg).* By G. NEWTON PITT, M.D. *Exhibited October* 28, 1904.

M. D., æt. 49. Nothing of importance in her family history. At birth a tumour to the left of the sternum was noticed. She has had numerous subcutaneous painless tumours so long as she can remember. In 1897 Mr. Golding-Bird removed a large unencapsuled fibrous tumour from the left calf, and in 1895 a large pendulous tumour, weighing 3½ lbs., was removed from the left side of her chest. In 1877 this was a pendulous pigmented mass five inches long. New tumours appear from time to time; at first they are firm and prominent, and gradually, as they increase in size, they become softer.

Present condition.—Her general health is good and all her organs are sound. There is a diffuse fibroid swelling over the left calf, forming a condition of elephantiasis mollis, with an irregular radiating mass of fibroid bands, the sequel of the old operation. The circumference of the left leg is 14½ and of the right 11¼ inches. There is a raised pigmented hairy mole on the right cheek, with molluscum tumours. A mass 4 inches by 1½ was removed in this region in 1877, and new tumours have grown since. There are innumerable raised tumours ¼ and ¾ inches across, scattered over the limbs and face, and to a less extent on the body. The skin over them is generally not pigmented. There are

numerous small pigmentary spots, freckles, on the limbs, and some large areas of pigmentation, which slowly increase in size. There is considerable deformity, with a projecting mass on the upper part of the sternum. There is a soft smooth fibrous mass pendulous from the left side of the hard palate, with an arrow base near the alveolus, occupying more than half the roof of the mouth. There is a tumour in the left lower eyelid, and four hard small masses in the palm of the left hand. Many of the tumours have appeared within the last five years. There is no evidence of any obvious tumours of the larger nerve-trunks. The case belongs to the group described by von Recklinghausen in 1882, in which neuro-fibromata, molluscum fibrosum, and cutaneous pigmentation are associated together, and tend ultimately to develop sarcoma. The bibliography and the whole question was fully discussed by Dr. Rolleston in the *Lancet*, July 29th, 1899.

I have been unable to find a record of any tumour growing from the hard palate of a size and similar nature to the one presented by this patient.

XII.—*A Case of von Recklinghausen's Disease.* By Charles R. Keyser. *Exhibited October* 28, 1904.

JOHN H., æt. 38½, has had all his life a number of small, rounded, subcutaneous tumours in various parts of the body, but the number has been and is slowly increasing. These tumours do not give rise to any symptoms whatever, and the patient complains solely of cough and shortness of breath. This cough has been present for nineteen years, but is much worse in the winter.

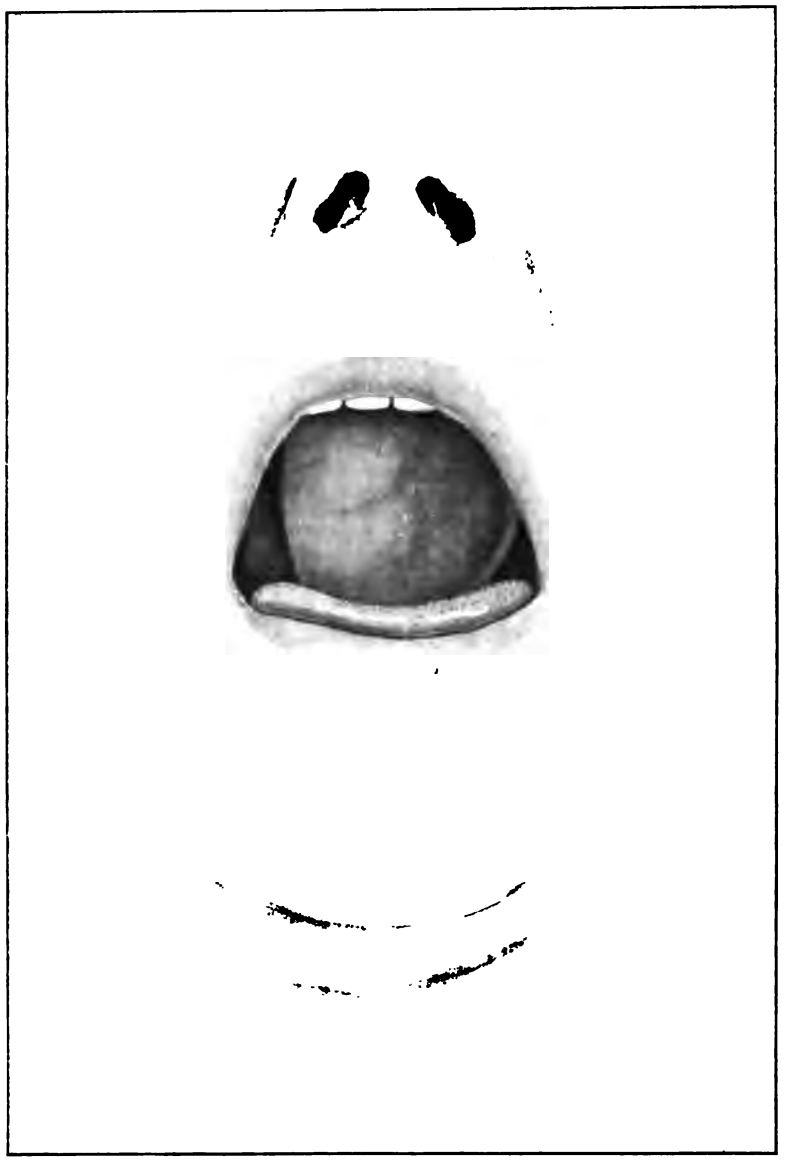
When 4 years old he had an attack of scarlet fever, which was followed by deafness. In 1891 he underwent an operation for piles, since which he has suffered somewhat from diarrhœa, with occasional incontinence of fæces. He has one brother who (he says) is affected with multiple tumours in a manner similar to himself, only more markedly so; the general health, however, is not affected. His mother is said to have died of the same disease; she had a large mass of growth extending across the front of her chest. The father died of ? phthisis.

The patient is a very short man, being only 4 feet 11 inches,

PLATE XII.

Illustrating Dr. G. Newton Pitt's paper on a case of Recklinghausen's Disease.

A large smooth pendulous fibroma can be seen hanging from the hard palate.



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is exceedingly deaf, and in consequence appears somewhat unintelligent. The face is very dark in colour, probably the result of chronic venous congestion from chronic bronchitis and emphysema. There are present a very large number of small, rounded, subcutaneous tumours, practically all over the body, the hands and feet excepted. These tumours are all soft in consistence and are absolutely painless. The largest are, one on the back of the left side of the head, one in the second right intercostal space, close to the sternum, one on the surface of the abdomen, right side, and one on the inner side of the lower part of the left thigh. Only the one on the abdomen, however, could be termed a molluscum fibrosum. The greatest profusion of nodules is seen on the back, close to the spinal column, and on the chest and abdomen. The arms and legs are only slightly affected. There is a small area of pigmentation present in the right hypochondrium, and another just below the inner end of the left clavicle. There are no signs of disturbances of the motor functions, of pressure on the spinal cord, or of malignancy in any of the tumours.

XIII.—*A Case of Lymphangiectasis, with Escape of Chyle.*

By A. E. Barker. *Exhibited October* 28, 1904.

A MAN, æt. 20, has a cystic swelling of varying dimensions above Poupart's ligament, and enlargement of the thigh. The disease has been noted since the age of 7 years. All over the inguinal region are small blebs of skin which can be emptied of fluid and which then refill at once. Chyle escapes from an opening in Scarpa's triangle as large as a crowquill. If the escape is temporarily prevented by a pad an enlargement occurs in the iliac region. Then the chyle, when allowed, escapes in a stream, and the patient feels relief from tension. If allowed to run, a pint or so escapes in a very short time—indeed, in a few minutes. The stream then slackens, and the fine, milk-white appearance gradually changes to a clearer one, and ultimately there is only a trickle of ordinary lymph from the opening. In this way between three and four pints have been collected within twenty-four hours. Towards the end he feels a kind of lassitude, and is glad to re-apply the pad firmly. Some

years ago the swelling in the inguinal region was mistaken in the country for a psoas abscess and incised. There was then some difficulty in getting the incision to heal. The general health is excellent.

XIV.—*Aortic Aneurysm. Innominate Aneurysm* (?). *Simultaneous Ligature of the Carotid and Subclavian Arteries.* By CHARTERS SYMONDS, M.S. *Exhibited October* 28, 1904.

JOHN G. B., æt. 44, was admitted into Guy's Hospital on August 8, 1904, under Dr. Hale White. By occupation an engine-fitter, his work at times is heavy, involving hammering and the lifting of machinery. There is no family history of inherited ailments, his father dying at 66 and his mother at 74. He is married and has three healthy children. He was born in Darlington. There is no history of any personal illness, and none of syphilis. He is a very healthy-looking man, well built, and temperate.

He states that he has had pain in his occipital region and on both sides of the head since October, 1903. On July 24 last he first observed a swelling in the neck while still in full work.

When admitted the condition was much as at the present time. A soft pulsating swelling appears above the sternum, reaching along the line of the carotid. It extends to the left of the median line also, and here it is possible to get the fingers under the margin. Along the line of the subclavian there is not any marked extension. There is dulness between the second intercostal space and adjacent part of sternum.

Before operation there was no bruit. Some difference of opinion existed as to the exact distribution of the aneurysm, whether it was aortic chiefly, extending up the neck, or whether it was mainly carotid. The involvement of the innominate seemed, from the position of the sac, likely. With the object of checking the extension, it was decided to ligature the subclavian and carotid arteries simultaneously, though no great promise could be held out. For this purpose he was transferred to the care of Mr. Symonds.

September 9, 1904, the operation was carried out, both

vessels being secured with thick floss silk. The ligatures were tightened till the distal pulsation ceased, the inner coats presumably not being ruptured. On the fifth day pulsation was observed in the radial artery, and a bruit was heard over the aneurysm, especially intense near the subclavian artery. Primary union followed.

The patient is exhibited to show that there is no change in the size of the swelling, and that no injurious effect has followed ligature of the vessels, also to call attention to the bruit. The appearance of the bruit after the ligation, and its point of chief intensity being towards the subclavian artery, suggest the loosening of the silk on this vessel.

XV. — *Bony Union, after Fracture of the Patella, without Operation.* By CHARTERS SYMONDS, M.S. *Exhibited October* 28, 1904.

F., æt. 37, exhibits a true bony union of a transverse fracture of the patella. He was admitted into Guy's Hospital July 18, 1901, under my colleague, Mr. Golding-Bird, with a transverse fracture, with slight separation. The skiagram taken at this time shows a quarter to an eighth of an inch separation, so that the case was a favourable one for treatment by splints.

The man broke the left patella, and was admitted again into Guy's Hospital, under Mr. Symonds, on August 15, 1904, with a well-marked separation of the fragments. The opportunity thus occurred of examining the condition of the right patella. The outline of the bone is complete, and no separation can be felt. The knee possesses its full use. The skiagraph shows the bone to be solid throughout, though somewhat altered in shape. The extreme rarity of this result, which cannot be explained solely by the small separation, is well known. The left patella has also been treated by splints. It is now rather more than two months since this second fracture, and the union here seems also to be osseous.

The second skiagram of this knee, made September 30, 1904, shows apparently the union to be bony.

It may be added that in both cases the fracture was of the transverse variety, and produced in the ordinary way.

XVI. — *Popliteal Aneurysm four and a half months after Ligature of the Superficial Femoral Artery.* By Mr. CHARTERS SYMONDS, M.S. *Exhibited October* 28, 1904.

H. D., æt. 37, was admitted into Guy's Hospital, under the care of Mr. Symonds, on June 11, 1904. On May 9 he noticed some pulsation after his work. By the end of May the leg swelled, and he sought advice of Dr. Hudson, of Cobham, who, recognising the aneurysm, sent the patient to the hospital.

On admission the popliteal space was occupied by a typical aneurysm, and the leg was one and a half inches larger from œdema. The aneurysm, judging by the increasing œdema, and the pain, was rapidly enlarging.

On June 17, the œdema of the leg having subsided a good deal, the superficial femoral was ligatured in Hunter's canal with silk. Size No. 2 was employed, and closed carefully so as not to rupture the coats. Primary union was secured, and the aneurysm began steadily to shrink. The man was kept in bed with great difficulty for four weeks, and was discharged on August 3.

At the present time a hard though small mass can be felt in the popliteal space. There is no pulsation to be felt in the distal arteries. The leg is not so useful as it ought to be, chiefly because the man declines to make any effort, and rather uses his ailments as an excuse for idleness. The foot is warm, sensation is good, and there is no œdema.

XVII. — *An Instrument for the Examination of the Rectum and Sigmoid Flexure (the Electric Sigmoidoscope).* By P. LOCKHART MUMMERY, B.C., F.R.C.S. *Exhibited January* 27, 1905.

THE instrument is a modified pattern of Strauss's sigmoidoscope or rectoscope.

The instrument can be used without fear of damage to the rectum, and no anæsthetic is necessary. In a normal individual it is possible to examine every inch of the mucous

membrane, from the anus to the middle of the sigmoid flexure, or further. The distance to which the tube can be passed depends almost entirely upon the mobility of the sigmoid; when there is a short sigmoid mesentery or extensive adhesions round the bowel the tube cannot be passed to its full length. In using the instrument the patient is placed in the genu-pectoral position, or in the left Sims' position. Perhaps the greatest value of the instrument is that it now makes it possible to diagnose cancer high up in the bowel before fixation or secondary gland enlargement has occurred, and at a time when removal can be attempted with comparative safety and with a reasonable prospect of success.

Cases.

Case 1.—The patient was a man, æt. 72. He was suffering from alternating diarrhœa and constipation, and the frequent passage of blood and slime by the rectum. Nothing could be detected on examining the rectum, and on examining him *per abdomen* under an anæsthetic, nothing could be felt. I then examined him without an anæsthetic, by means of the sigmoidoscope, and was able to see a growth in the last loop of the sigmoid flexure. I was further able to ascertain that it was situated on the mesenteric side of the bowel, and was movable. This patient was subsequently operated upon, and the growth was found to be exactly as it had been seen with the sigmoidoscope.

Case 2.—The patient was a man, æt. 62. In this case the growth could not be felt, but an examination with the sigmoidoscope revealed a growth in the centre of the sigmoid flexure.

Case 3.—The patient was a man, æt. 49. He was suffering from symptoms of rectal cancer, but on examining him in the ordinary way *per rectum*, with the finger nothing could be felt. On getting him to strain down it was just possible to feel the growth, but not to examine it properly. With the sigmoidoscope the growth could easily be seen, and it was found to be very extensive. I came to the conclusion that it could only be removed by the abdominal route with great difficulty. The patient decided, I think wisely, not to have anything done.

Case 4.—The patient was a man, æt. 53. He was a patient of Mr. Dent's, at St. George's Hospital. There were symptoms of a growth in the sigmoid flexure, and it was thought

that a growth could be felt in the left iliac fossa *per abdomen*. With the sigmoidoscope it was possible to ascertain that the growth was firmly fixed in the left iliac fossa. The appearances immediately below the growth are shown in the drawing. No operation was performed.

Case 5.—The patient was a man, æt. 63, who had been suffering from symptoms of a growth in the rectum for eighteen months. On examining him with the sigmoidoscope a large papillomatous growth could be seen situated on the posterior wall of the rectum, just above the middle Houston's valve, as shown in the drawing. This growth could also be felt with the finger.

I subsequently removed the growth, which was a papilloma about the size of a five-shilling piece. The coccyx was removed and the growth was excised, half an inch of healthy mucous membrane being taken out all round it. The wound in the rectum was closed by sutures, and the skin wound was also closed, except for a drainage-tube. The patient made a good recovery, and left the hospital in three weeks. There was only slight narrowing of the bowel at the spot where the growth had been removed.

Case 6.—This is a case of an ulcer of the rectum, just above the upper Houston's valve, in a woman who had an osteo-enchondroma of the sacrum.

Case 7.—This was a case of chronic ulcerative colitis of two years' standing, under the care of Dr. Ogle. Fine granulations could be seen all over the mucous membrane, and there were numerous ulcerated patches.

Case 8.—The drawing shows the appearance of the mucous membrane in a case of gonorrhœal proctitis, in which the inflammation had spread up into the sigmoid flexure.

XVIII.—*Case of Extensive Syphilitic Necrosis of Skull, with Intra-cranial Abscess, but with a complete absence of Cerebral Symptoms.* By J. Hutchinson, Jun. *Exhibited January* 27, 1905.

J. H. W., at age of 18, contracted syphilis, treated only for a short time, in the Army.

Four years later had rupia and severe joint-pains; anti-syphilitic treatment was resumed.

PLATE XIII.

Illustrating Mr. P. Lockhart Mummery's Instrument for the Examination of the Rectum and Sigmoid Flexure (the Electric Sigmoidoscope).

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Bale & Danielsson, L[td] lith.

No further symptoms for ten years, until September, 1904; then a lump formed on vertex of head, which burst after four weeks, many sinuses formed, as well as a large circular area of necrosis, over which scalp was entirely lost. Free suppuration continued, the pus working up through the dead bone and pulsating with the cerebral pulse. In left eye posterior synechiæ from iritis; this is the only other sign of syphilis.

He was trephined through the dense dead bone, the inner surface of which was found to be eroded, there being much pus between the dura mater and bone.

Is now taking two to three drachms of iodides daily. Has not had the slightest cerebral symptom, neither headache, giddiness, nor impairment of memory. Until his admission to hospital on December 28th, he continued to do his work as a letter-carrier without any difficulty.

NOTE.—*April* 15, 1905. The large sequestrum is not yet loose, and suppuration is still profuse beneath it.

XIX.—*Ureteral Calculus in a Girl aged 7. Ureterotomy.*

By C. H. FAGGE. *Exhibited January* 27, 1905.

A. C., æt. 7, came under my care at the Evelina, in 1901, for hæmaturia and pain in the right loin, which resisted medicinal treatment and rest. In 1903 she was admitted for the same symptoms into another hospital, and an umbilical hernia, which was thought to be the cause of her pain, was operated upon. In August, 1904, she again became an out-patient, under Mr. Sidney Scott, at the Evelina; she then had abdominal pain, referred to the right side, which, when acute, caused sickness, and her urine, which was smoky, contained red corpuscles and albumen. She was admitted on September 14 with pain on the right side of the abdomen, just above the level of the umbilicus, extending into right iliac region. Nothing abnormal could be felt, and her urine was then normal, except that it was offensive and contained bacteria.

The X rays discovered nothing abnormal, but there was a tender spot opposite to and two inches to the right of the umbilicus.

Under an anæsthetic, a small, hard mass was felt to roll

under the finger at a spot corresponding to the position of deep tenderness.

On October 6 the right kidney was exposed and palpated from the front, and appeared normal, but a small calculus was felt in the ureter about one inch below the lower end of the kidney; the ureter was incised and the calculus removed; the ureter and the layers of the abdominal wall were sutured with catgut. A trace of albumen was noticed in the urine for a week after the operation, from which she made an uneventful recovery.

XX.—*Localised Flushing and Sweating of the Cheek on Eating, which, together with "Striæ patellares," followed Appendicitis ten years ago.* By F. Parkes Weber, M.D. *Exhibited January* 27, 1905.

THIS patient, now æt. 26½, was shown by Dr. Weber at the Clinical Society on October 22, 1897. The main symptoms are still present, and there are some other points to be mentioned. When he eats anything solid (for instance, a piece of sugar or an apple) a patch of redness appears almost immediately on the left side of the face as if he had received a sharp blow on the cheek. The reddened area is very hot, and sweating quickly follows. Occasionally a much smaller area of redness and sweating appears on the right side of the face in front of the lobe of the ear, when the phenomena on the left side are at their maximum. The swallowing of fluids, such as milk, etc., does not cause the flushing and sweating to appear, but the rolling of a little vinegar and water about in the mouth, without biting or swallowing, calls forth the curious reaction fairly promptly. The reaction commences more quickly when he uses his teeth on the right side for biting his food than when he uses those on the left side. By merely opening and shutting his jaws or chewing a piece of wood a very slight degree of redness is all that can be produced. The flushing and sweating subsides two or three minutes after he leaves off eating, but it takes a longer time for the redness to completely disappear. It was found that the phenomena were somewhat less marked when a cocaine lozenge was taken before his food, but the

use of these cocaine lozenges has not been encouraged. A similar diminution in the symptoms followed treatment by bromide and belladonna (ten minims of the tincture three times daily) internally, and belladonna liniment externally.

In the present case there is no neuropathic family history. The patient suffered from appendicitis in December, 1894, and the appendicitis was followed by parotid buboes on both sides of the face, which had to be opened, and the scars of which still remain. About March, 1895, he was operated on for the appendicitis, and it was after that operation that the flushing and sweating of the face were first noticed. Dr. Weber believes the most probable explanation of the phenomenon to be that some sympathetic nerve-fibres are involved in scar-tissue on both sides, but chiefly on the left side, and are stretched or pulled on owing to the physiological swelling of the salivary glands during eating. There is very slight diminution of tactile sensation in the neighbourhood of the scars. No localised sweating or flushing occurs when the patient becomes hot by exercise or excitement, and there are no pupillary or other phenomena indicating any involvement of the rest of the cervical sympathetic. The patient's general health is now good. The appendicitis scar is healthy, and there is no sign of present disease of the thoracic and abdominal viscera or of the central nervous system. The diminution of hearing on the left side is due to old middle-ear disease. The knee-jerks are natural.

The rather uncommon atrophic transverse scars in front of this patient's knee-caps and just above them, known as *striæ patellares*, are peculiarly well marked and long. They were first noticed after the appendicitis ten years ago.

XXI. — *A Case of Goitre in a Child æt.* 5. By Charles R. Keyser. *Exhibited January* 27, 1905.

THE patient is a boy, æt. 5, who has had a lump in the neck since September, 1904. When first noticed, other symptoms, *e.g.* anorexia, occasional vomiting, and a feeling of malaise, were present. The tumour, which presented all the signs of a parenchymatous goitre, gradually increased in size until it was as large as a hen's egg.

The boy has always lived in London; there is no history of a similar condition occurring in any other member of the family.

The treatment has consisted in the administration of small doses of tincture of iodine and potassium iodine internally and the local application of biniodide of mercury ointment (gr. vj–℥j). The result has been a rapid diminution in the size of the tumour.

XXII.—*Case of Acromegaly.* By H. Batty Shaw, M.D. *Exhibited January 27, 1905.*

H. W., æt. 36, a bricklayer, sought relief at University College Hospital for numbness and tingling of the hands, from which he had suffered on and off for three years. The hands and fingers have been noticed to be large for four or five years; there is no increase in the length of the fingers, and the enlargement of the hands does not extend beyond the wrists. The lines in the palms are much deepened. Eight or nine years ago the size of the boots worn by the patient was No. 6, now it is No. 8. The feet and toes are large, especially the great toe; the skin over the heels is thick and tough. There is no kyphosis. The lower lip is gradually becoming larger and projects more than formerly. The hair is rather coarse and grows very quickly. There appears to be some enlargement of the scapulæ and ribs, and the patient has noticed that recently he has had to increase the size of his hat. His expression is somewhat heavy; the subcutaneous tissue of the eyelids is a little thickened, and the nose broadened; the wrinkles on the face and forehead are exaggerated; the tongue is enlarged, and the voice is hoarse and deeper in pitch than formerly; the muscles are not wasted. The discs and fields of vision were found by Mr. Percy Flemming to be quite normal. Besides the above-mentioned acroparæsthesia, the patient is occasionally troubled with headache; formerly it persisted for several months continuously; lately he has noticed an increasing drowsiness after meals, otherwise the mental condition is quite normal. His appetite is rather excessive; the urine is quite normal. He has only one child, æt. 5 years.

Radiogram (by E. S. Worrall, Esq.)—This shows the fan-like arrangement of the metacarpal bones, and possibly some slight increase in width of the first phalanges; there are no exostoses.

XXIII.—*Case of Acromegaly occurring in a Patient suffering from Chronic Chest Disease.* By H. Batty Shaw, M.D. *Exhibited January* 27, 1905.

Mrs. F., æt. 29, was formerly an out-patient at the Brompton Hospital for Consumption and Diseases of the Chest, under the care of Dr. Horton-Smith Hartley, and is now an in-patient under Dr. Habershon's care.

The hands and fingers are enlarged, more especially shown by the broadening of the metacarpal region and increase of girth of each digit; the patient's ring can no longer be readily removed. There is slight thickening of the terminal parts of the fingers, and the nails, in the case of some digits, appear to be slightly coarser than usual. The lines of the palms are distinctly deepened, the wrists and forearms are not involved. Six or seven years ago the patient wore boots of No. 5 size; now she wears No. 6. The feet and toes are enlarged. The malar eminences are slightly prominent; the lower lip protrudes; the teeth are not abnormally separated; the tongue is distinctly enlarged. The hair is coarse and long. The eyelids and skin generally are a little thick and swollen, and the features coarser than formerly (see photographs). The expression is dull and heavy. There is no notable alteration in the voice. The optic discs and fields of vision, examined by Mr. Percy Flemming, are normal. The patient has been frequently troubled with headache during the last five years; vomiting in the morning has occurred on and off for three years, and insomnia has been noticed during the same period; the patient is somewhat dull mentally. The urine is normal. The appetite is excessive. The catamenia are now normal, but a period was missed in June, 1904. There has been one miscarriage, and this was succeeded by the birth of a child three and a half years ago; there are no other children.

Radiograms.—These were prepared by Dr. A. H. Greg. The radiogram of the hands reveals slight changes similar to those

noted in the phalanges of the preceding case. The radiogram of the chest shows that the disease is probably limited to the left pleura above and external to the heart; the extreme left apex appears free from disease. The radiogram of the head

FIG. 2.—PHOTOGRAPH OF MRS. F—, ÆT. 22 YEARS.

shows the outline of the pituitary fossa, which does not appear to be enlarged.

The lesion in the left lung is of an obscure nature. There is dulness to percussion of the upper part of the front of the chest, as well as reduction of vocal resonance and breath sounds.

There are very few adventitious sounds; the maximum impulse of the heart is drawn upwards and a little outwards. Somewhat similar signs are observable at the extreme upper part of the left lung behind. There is a history of left pleurisy; so far tubercle bacilli have not been found in the expectoration.

FIG. 3.—PHOTOGRAPH OF MRS. F—, ÆT. 29 YEARS.

The existence of this condition points to a possible explanation of the slight clubbing observed in some of the fingers; the character of the enlarged hands and the involvement of the face suggest that this is a case of acromegaly, and not of pulmonary hypertrophic osteo-arthropathy; the slight clubbing of the fingers, which is not a character of acromegaly, is explainable by the condition of the lungs.

XXIV.—Case 3. *Bilateral Wasting of the Subcutaneous Tissues of the Face in a Boy æt. 10.* By H. Batty Shaw, M.D. *Exhibited January* 27, 1905.

L. W., æt. 10, an only child, was brought to the Hospital for Consumption, Brompton, for what the mother feared was consumption of the lungs, because of the wasting of the child's face. This wasting has been more particularly noticed since an attack of measles two years ago, but the child attended the Victoria Hospital for Children for wasting as long as three years ago. At two and a half years of age there was apparently no wasting, as seen from a contemporary photograph. There is no history of a similar condition in any other member of the family; the boy's mother has had rheumatic fever twice, and the patient himself has had "growing pains," and occasionally shows choreic movements.

The wasting is confined to the face and is bilateral; it does not appear to involve the skin proper, or muscles or bony tissues, but would appear to affect the subcutaneous tissues.

There is no wasting elsewhere of the body and no signs of involvement of the nervous system; the muscles of the face are not paralysed, and sensation, etc., is normal.

XXV.—*A Boy three years after Removal of a Large Cystic Adenoma of the Left Kidney.* By W. G. Spencer, M.S. *Exhibited January* 27, 1905.

IN the summer of 1901, when the boy was æt. 12, the mother first noticed the swelling, but she said he had always had a "big stomach." In January, 1902, the boy was first taken to Dr. Redmond Roche, who diagnosed the case. Abdominal nephrectomy was done by a T-shaped incision; some vascular adhesions to the diaphragm caused a temporary disturbance of respiration. The half of the specimen in the Westminster Hospital Museum is shown, and Mr. Shattock's description of the other half in the College of Surgeons Museum is appended. The patient has grown much and is in robust health.

XXVI.—*A Child after Recovery from Infantile Paralysis of the Deltoid.* By W. G. SPENCER, M.S. *Exhibited January* 27, 1905.

A GIRL, æt. 4½, was attacked on September 15 with pain in the right arm, and the next day the arm was paralysed. On September 21 a marked reaction of degeneration was found affecting the deltoid, triceps, and biceps.

When the subject of nerve-grafting for such cases was before the Society in October last, the girl was in Dr. de Havilland Hall's ward, and could not then raise the arm from the side at all. There were then only movements in the forearm and hand. The child has been kept under Dr. Hall's observation, and has had the arm rubbed and electricity used. She can now raise it easily above the head and seems to be recovering well.

XXVII.—*Case of a Child with Graves' Disease and Myxœdema (?)* By F. E. BATTEN, M.D. *Exhibited January* 27, 1905.

A. A., æt. 10, eldest of family of six children; paternal uncle died of insanity.

History.—Thirteen weeks ago fell down flight of stairs. Eleven weeks ago frontal headache, and of such severity that the child has been kept in bed. Vomited on one occasion only. Six weeks unable to walk, and staggered when walking. Appetite large. Face and hand noticed to be puffy. The throat has become large lately.

Present condition.—Fat child; pale, puffy face; squeaky voice; placid, slow manner; considerable enlargement of thyroid gland; heart rapid; pulse 118–140; no proptosis; walks in peculiar waddling manner; no evidence of disease of the nervous system; no evidence of visceral disease except points above mentioned. The child took thyroid gland tabloid for several weeks without any improvement and was in the same condition when seen three months later.

XXVIII.—*Case of Xanthoma Diabeticorum.** By Sir Dyce Duckworth. *Exhibited January* 27, 1905.

GEORGE M., æt. 23, a clerk. Diabetes since September, 1904. Under treatment from that time by private doctor, then at St. Thomas's Hospital till November, 1904, when he discharged himself. Admitted St. Bartholomew's, December, 1904.

Family history.—A sister died of diabetes, æt. 31.

December 24.—Over both elbows a large number of small hard papules have appeared, shotty and somewhat painful on pressure; a small amount of yellowish inspissated material can be pressed out of them.

December 31.—Papules over elbows increased in size and fresh spots have come at elbows, and also on back, buttocks, neck, face (scanty), and lower extensor surface of knees. Eruption is symmetrical.

January 25.—Papules increasing in size and number.

Amount of urine passed: average 100 ounces in twenty-four hours; s.g., 1034. Sugar, on admission, 25 grains per ounce; January 25, 12 grains per ounce. Body weight, on admission, 8 st. 6 lb.; January 25, 7 st. 8 lb. Iron perchloride reaction present.

XXIX.—*A Case eighteen months after Fractures of the Scaphoid, Semilunar and Cuneiform Bones of the Carpus.* By Edred M. Corner, F.R.C.S. *Exhibited January* 27, 1905.

EIGHTEEN months ago a young man, A. K., a printer, had his hand caught in the machinery and twisted. He went to a hospital and had the arm put into splints, in which it was kept for a month. He was told that the injury was a dislocation. Seven weeks after the accident he returned to work. He now complains that the hand is weak, occasionally painful, and has lost some of its power of movement.

On examination, the wrist is seen to have all its movements considerably limited. The two styloid processes are more or less on a level with each other. An irregular bony prominence can be felt in front of the wrist, and a similar

* For fuller details of this case see page 183.

prominence can be felt behind over the distal portion of the carpus. It will also be noticed that the "anatomical snuff-box" is practically obliterated, whilst a new one has been formed between the extensor longus pollicis and the extensor minimi digiti.

An examination of the skiagraph, made by Mr. Greg, shows, first of all, that the semilunar bone has been more or less pulverised and the fragments displaced forwards, helping to form the bony prominence previously referred to. The scaphoid has been smashed and displaced in a similar way, but the most radial portion of it has remained more or less in its normal place. These two bones have borne the main brunt of the violence and have suffered most. The cuneiform bone has had the portion nearest the radius broken. This damage has obviously been done by the lower end of the radius being forcibly driven on to the carpus. This is shown by the close proximity of the ulna to the cuneiform. The distal row of the carpal bones has been displaced backwards, forming the prominence which can be felt in that situation. Although the man is over 20 years of age, the lower radial epiphysis does not appear to have joined to the shaft. The man's hand would doubtless have retained more of its powers had it been taken out of the splints and treated by massage. Eighteen months have now passed, so that there is little hope of improving the condition much.

XXX.—*Epileptiform Neuralgia limited to the Right Superior Maxillary Nerve. Intra-cranial Resection (through the temporal fossa) of the Superior Maxillary Division, on February* 27, 1903. By J. HUTCHINSON, Jun. *Exhibited January* 27, 1905.

THE case is reported in Vol. XXXVII of the *Clin. Soc. Trans.* (1904), p. 136. The patient had suffered for six years from severe spasmodic neuralgia involving the right cheek and upper jaw. Whilst the pain continued to be limited mainly to the superior maxillary division, there were slight radiations of it to the forehead and lower jaw. These have been entirely cured by the operation on the second division.

At the time the case was reported, one year after the operation, some little doubt was expressed as to the prospect of recurrence. The patient has continued to serve on board a Cape liner, and has therefore constantly been exposed to wind, rain, and changes of temperature. He has not had the slightest recurrence of neuralgia, and, owing to his being able to eat in comfort, has put on weight considerably. Before the operation taking food invariably brought on epileptiform attacks. The anæsthesia of the cheek gives him no trouble.

XXXI.—*A case of Anæmia Splenica Chronica.* By J. H. Bryant, M.D. *Exhibited February* 24, 1905.

Arthur C—, æt. 48, a man who has always been temperate, and has never been abroad, was admitted into Guy's Hospital under my care on January 25, 1905, for pain in the abdomen and hæmatemesis. He was born at Northampton, and lived there until he was 11. In 1868 he came to London, and has resided there off and on ever since. After leaving school he entered the London, Chatham, and Dover Railway fitting-shop, and worked there until 1900. Measles and whooping-cough in childhood are the only illnesses from which he has suffered. In July, 1876, whilst at work, he fell into a dry pit, striking the left side of his abdomen against an iron rail, and so severely injuring himself that he was taken to St. George's Hospital, where he was told that he had ruptured his liver (? spleen). He states that his abdomen was enormously distended, and that he was unable to pass his urine naturally. He remained in the hospital for a month, but was unable to resume work until six months later, when he noticed dyspnœa, giddiness, and pain on micturition. In 1900 he complained of acute abdominal pains, and on consulting his doctor was told he was suffering from an enlarged spleen. Ever since the accident he has been subject to bilious attacks.

In January, 1904, he had a severe attack of vomiting and hæmatemesis for the first time. On January 14, 1905, he was again suddenly taken ill with severe abdominal pain, vomiting, and hæmatemesis.

He has had neither syphilis nor gonorrhœa. He has always

been of temperate habits. He has suffered from constipation all his life.

He states that his fingers have been clubbed ever since he can remember, for when he first commenced work the men used to remark on their curious shape. About a year ago he first noticed a curious discoloration of the skin covering his feet and legs. The yellowish appearance of the skin of his face has been noticed all his life, but became more pronounced after the accident.

Condition on admission.—Pulse, 80; temperature, 98°; respiration, 26. The skin of his face and neck has a curious sallow, yellowish-brown tint more like sunburn that jaundice. The sclerotics are not pigmented. There are a few dilated venules over his malar prominences. The face has a somewhat pinched and wasted appearance. There is very marked clubbing of the fingers and toes. There are several irregular-shaped patches of brownish-yellow pigmentation of the skin covering the dorsum of the feet and the extensor surface of the legs. There are also patches of ecchymosis fading insensibly into the areas of pigmentation, which suggest that a hæmorrhage into the skin is the beginning of the pigmentation.

The spleen is considerably enlarged. Its surface is smooth and hard. Two distinct notches can be felt in its anterior border, one just below the costal margin, the other just below the level of the umbilicus.

Urine 1020, acid, of normal colour; no bile, blood, albumen, or sugar present.

The blood.—Red blood corpuscles, 4,500,000 per c.mm.; hæmoglobin, 80 per cent.; colour index, 0·88; white blood corpuscles, 2500 per c.mm.

Differential count of leucocytes.—Small lymphocytes, 22·5 per cent.; large lymphocytes, 5·5 per cent.; polymorphonuclears, 69·0 per cent.; coarsely granular eosinophiles, 2·0 per cent.; myelocytes (eosinophile), 0·5 per cent.; basophiles (doubtful), 0·5 per cent.

No nucleated red cells. All the red cells were of good shape, natural size, and stained well.

XXXII.—*A case of Unilateral Dislocation of the Atlas from the Axis, with Fracture of the Anterior Arch of the Atlas.* By EDRED M. CORNER, M.B., B.C. *Exhibited February* 24, 1905.

A MAN, æt. 21, fell off a horse upon his forehead. He "saw stars," but was able to mount and ride home. A fortnight later the patient's neck was found to be rigid, and he carried his head a little flexed and turned to the right. He had a difficulty in opening his mouth, also in articulation and in mastication, but not in swallowing. The left transverse process can be felt between the mastoid process and the angle of the jaw; in place of the process on the right side the examining finger finds a depression, whilst at the back of the pharynx on the right side there is a prominence. A skiagraph shows the rotatory dislocation of the atlas from the axis on the right side, and also a fracture of the anterior arch of the atlas. The condition of the odontoid process is doubtful.

XXXIII.—*A case of Hysterical Blindness and Narcolepsy.* By PURVES STEWART, M.D. *Exhibited February* 24, 1905.

A MAN, æt. 22, previously healthy, had an attack of influenza six weeks ago. A fortnight ago he complained of pain in the left side of the chest, but with no abnormal physical signs, and he had insomnia for four or five nights. He was then given fifteen grains of sulphonal, and slept for nineteen hours continuously. Since then he has had attacks of sudden sleep, occurring at meals, during games, etc., lasting several hours at a time. Four days before admission he developed convulsive phenomena (opisthotonos, etc.) just before waking. Three days before admission he awoke to find himself practically blind. He can now only distinguish light from darkness, and the gait is that of a blind man. But he keeps the eyes directed habitually straight forward. Optic discs, pupils, and cranial nerves normal. No headache nor vomiting. Urine normal.

XXXIV.—*A case three Years after Muscle-grafting for Paralysis of the Left Upper Extremity.* By A. H. TUBBY, M.S. *Exhibited February* 24, 1905.

IN June, 1901, the boy, then æt. 10, was seen with infantile spastic paralysis of the left upper extremity. The forearm and hand were in the position of excessive pronation, with flexion at the elbow and wrist. The fingers were strongly flexed. An operation was performed two years ago. The pronator radii teres was detached, and passed behind the radius and re-attached so as to act as a supinator. The palmaris longus and other flexor tendons at the wrist were divided. The patient has regained extension at the elbow, supination of the forearm, and flexion and extension of the fingers.

XXXV.—*Primary Sore on the Eyelid of a Child, æt.* 3. By WILLIAM TURNER, M.S. *Exhibited February* 24, 1905.

A GIRL, æt. 3, had been quite well until early in January, when, without obvious cause, a pimple formed on the margin of the right lower eyelid towards the outer side, and has since steadily enlarged. An enlargement of the glands below the jaw on the right side was first noticed on February 11th, and there has since been a steady increase in size. The child has seemed ill since February 18. She had been sleeping with her mother. The mother, æt. 29, has had several living children, and two born alive who died after a few weeks, and one miscarriage. She was under treatment in August, 1904, for secondary lesions, mucous patches in the throat with enlarged glands, and an eruption on the hand.

XXXVI.—*A case of Lymphatic Leukæmia with microscopic Specimens of the Blood, showing an enormously increased number of white Blood Corpuscles.* By J. G. SLADE, M.D. *Exhibited February* 24, 1905.

THE patient, a house-painter, æt. 49, has been in hospital under Dr. Allchin. He has always been a teetotaller, without venereal history, and is a moderate smoker. He began to complain in the autumn of 1902 of weakness and pains all over, and lumps in the neck were first noticed by his wife. In May, 1903, a doctor found lumps in the neck, axillæ, and groins, larger then than they are now. He improved under arsenic, but before admission lost flesh, was short-winded, and his face became yellowish. He has had no bleeding nor diarrhœa, nor œdema of the ankles. He was found to have enlarged glands, supra-clavicular, axillary, and inguinal; they were soft, discrete, movable, and not tender. The liver extended two inches below the costal margin, the spleen was enlarged, extending two inches below the umbilicus, and the notch could be felt. His urine had a trace of albumen.

Blood examination, February 21st: red corpuscles 2,430,000; hæmoglobin 24 per cent.; white corpuscles 1,300,000; small 96 per cent.; large 3 per cent.; polymorphic 1 per cent.; of the polymorphic 5 corpuscles were finely granular to 1 coarsely granular; blood platelets scanty.

XXXVII.—*A case of Ossifying Sarcoma of the Flexor Longus Digitorum Muscle.* By J. HUTCHINSON, Jun. *Exhibited February* 24, 1905.

THE patient, a man æt. 35, has observed the tumour during the last two years. It overlaps the tibia, and extends from a hand's-breadth below the knee to close above the ankle. The radiograph indicates that the tumour is connected with the fibula, there being a large amount of bony radiations from it. The tumour interferes but little with the use of the limb, but has caused a certain amount of pain. The duration is at least two years. Pulsation in both anterior

and posterior tibial arteries does not seem to be altered at the ankle. The patient has sustained no injury to the part, and no cause can be assigned for the development of the tumour. He had secondary syphilis fourteen years ago.

Note.—April, 1905. The case was exhibited as one of tumour of bone, and the general opinion was expressed that amputation would be necessary. However, on exploratory incision, it was found that it involved the flexor longus digitorum muscle alone. It was almost entirely bony in structure, having but traces of sarcoma tissue at the margin. Being under the deep fascia, and in part the soleus, it was so bound down as to seem fixed to the fibula. The radiograph was remarkably deceptive in this respect. The patient recovered, with a very useful leg, being able to walk perfectly well.

XXXVIII.—*A Case of Disease of both Knee-joints, for Diagnosis.* By W. H. Battle. *Exhibited February 24, 1905.*

A MAN, æt. 37, in 1902 was attacked with pain in the calves, ankles, and feet, and after treatment under several practitioners, including the administration of salicylates and iodide of potassium, he had effusion in the wrist, elbow, and knee-joints. At the end of 1903 the effusion in the knees had become more marked. The right knee was aspirated; it did not improve for a time, but at present is less swollen, whilst the effusion in the left knee has increased. As he cannot follow his occupation he has come into hospital for treatment. Both knees are swollen and appear distended with fluid, but there is no complaint of pain or sign of inflammation. The outline of the joint is clearly shown, and margin of swelling upwards very definite. The swelling feels pulpy, is elastic, and decidedly lobulated. There is probably some fluid in left knee, but this is not certain. The condition is regarded as gummatous, but there is no history of syphilis.

XXXIX.—*Cases of Congenital Dislocation of the Hip, after Treatment by Manipulative Operation and Exercises, etc.* By J. Jackson Clarke, M.B. *Exhibited April* 28, 1905.

CASE I.—Amy T., the same patient whom I presented to the Society on October 28, 1904. At that time, one year and two months after operation, the head of the femur was felt in the groin, the joint was stiff and somewhat painful, and slightly flexed, the thigh abducted to 45°. The case was an example of the *rigid* variety of the pubic immediate result of the manipulative operation. As the result of patient's having done the appropriate exercises since she was last shown to the Society the head of the femur has receded to its normal position, the joint moves freely and without pain, both actively and passively; the flexion has been almost entirely removed, and the abduction reduced to about 10°, beyond which it would not be wise to reduce it, save gradually by the natural effects of walking. Patient can walk well without limping, save that the abduction causes her to *rise* on the affected limb.* As the head of the femur receded the trochanter rose to about ¼ in. above Nelaton's line. This is probably due to some alteration in the form of the acetabulum and of the neck of the femur. The restored joint is quite firm.

Case 2.—Phyllis —, æt 6½, when operated on in August, 1903, for congenital dislocation of the right hip with 1¼ in. shortening, and marked telescopic movement, and adduction, etc., patient walking with marked limp. Confirmed by skiagraph. Patient had never walked properly, and had attended a hospital for three years without benefit.

The primary plaster case was removed in February, 1904, and patient was allowed to walk; since there was little or no rigidity, a metal instrument was applied to maintain 60° abduction; at this time the head of the femur was internal to femoral artery. In June, 1904, patient was allowed to walk without an instrument, the diminution of abduction being left to Nature. January 17, 1905, the result appeared to be perfect, patient walking well, trochanter in Nelaton's line. A skiagraph confirmed the opinion that the head of the femur was in the acetabulum. At the present time this apparently perfect result is maintained.

* Owing to the slight degree of abduction referred to above.

On March 20, 1905, some pain was complained of, so the joint was manipulated under ethyl chloride, and patient walked away from the hospital, and has been free from pain since then. At the present time the patient walks well; there is only a slight rolling on both sides,* and the joint appears to be normal. A skiagraph has recently been taken, and was shown at the meeting.

XL.—*Case of Congenital Dislocation of the Right Hip-Joint, replaced by Lorenz's Method, December*, 1899. By T. H. Openshaw, C.M.G. *Exhibited April* 28, 1905.

D. P., æt. 13. First came under my observation at the London Hospital in October, 1899, at the age of 7.

The mother brought her because she limped. She could walk about all day, but always with a limp. She was two years and three months of age before she began to walk. No deformity was noticed before that time. Subsequently the mother noticed that the right buttock was broader and flatter than the left. After the age of five the deformity and shortening and limping rapidly increased.

When first seen in October, 1899, there was a shortening of the right leg of an inch and a quarter. All the movements of the hip-joint were normal, except abduction and rotation out, which were both limited. Flexion was exceedingly free. The head of the bone could be distinctly felt on the dorsum ilii.

She was admitted on November 7, 1899, and forcible extension by means of pulleys, and manipulation of the joint, with tenotomy of the adductors and tensor vaginæ femoris, was performed.

The child was kept in bed, and continuous extension by means of weights was kept up for three weeks. A fortnight later another attempt at forcible reposition by manipulation was made, but without success.

On December 21, 1899, the hip was replaced by Lorenz's method after prolonged manipulation, and the leg was put up in plaster of Paris in a fully abducted position. The leg was

* This is due probably to a slight degree of coxa vara and has no relation to the previous marked sinking on the affected limb.

altogether in plaster for two years and five months, the child being allowed to walk with the leg in the fully abducted position after the first plaster had been on six weeks, so that continual pressure was exercised upon the acetabulum by the head of the femur. During the past three years she has been able to walk about as well as other people, going to school, moving about quickly, jumping, etc.

April 28, 1905.—The condition at this date is as follows: The left leg measures $30\frac{7}{8}$ inches, the right leg $29\frac{1}{2}$; the left tibia is 13 inches and the right tibia is $12\frac{3}{4}$ in., the left femur $15\frac{7}{8}$ in., the right femur $15\frac{1}{2}$ in. There is thus a difference of $\frac{3}{8}$ inch in the two femora, and $\frac{1}{4}$ of an inch between the two tibiæ (due to atrophy from imperfect use), in all $\frac{5}{8}$ of an inch, so that the shortening due to displacement at the hip-joint is really only $\frac{3}{4}$ of an inch. This is confirmed by Bryant's triangle, the base of which on the left side is $2\frac{1}{2}$ inches, and on the right side $1\frac{3}{4}$ inches.

Nelaton's line also shows the upper border of the right great trochanter displaced upwards $\frac{3}{4}$ of an inch.

The hip-joint can be flexed to a right angle, and hyper-extended to the amount of ten degrees; abduction is possible to an angle of forty degrees, adduction to the extent of a normal joint, rotation out to an angle of fifteen degrees, and rotation in to an angle of fifteen degrees.

The head and neck of the bone are rotated out upon the point of attachment of the neck to the great trochanter. The surface of the head of the bone thus looks forwards and inwards when the internal condyle is directed inwards. The head of the bone can be felt in a line with the anterior superior spine, at a point two inches below it. The head of the femur therefore cannot be said to rest exactly upon the acetabulum. The leg is held in a normal position as regards rotation out and rotation in. The appearance of the top of the thigh is normal, except for a slight fulness at a distance of about two inches from the crest of the ilium, and a depression corresponding with the gap in the adductors, the result of their division by tenotomy.

The child still walks with a slight limp, but this is mainly due to shortening of the leg. There is no telescopic movement of the hip-joint, and the hip can be pressed firmly up without pain or discomfort.

This case is a typical example of a large percentage of the results of Lorenz's method of replacing a congenital dislocation of the hip. The head of the bone is not perfectly in

joint, and does not rest upon the acetabular cartilage. It is displaced outwards and forwards so that there is a fulness on the front of and external to the centre of the joint, and a displacement outwards of the whole bone, making the great trochanter more prominent than on the opposite side. The firmness of the joint, however, and the joint movements, are as perfect as can be desired, although the anatomical reformation of the joint is not complete. In my opinion in very many cases it is practically impossible to put the head of the femur upon the acetabulum and keep it there for a time sufficiently long to secure the dilatation of the narrow portion of the capsule of the hip-joint, which always intervenes between the head of the femur and the acetabular cartilage, when the femoral head is first replaced.

Until this narrowed portion of the capsule has been dilated, it is impossible for the head of the bone to rest directly upon the cartilage of the acetabulum, and hence it is that many of the cases treated by Lorenz's manipulation result in an anatomically imperfect hip-joint, though functionally the joint may be perfect.

This liability to incomplete reposition and subsequent partial reluxation is accounted for in my opinion by two facts, first, the almost complete impossibility of dilating the narrowed portion of the capsule, and second, the presence of a rotation (generally forwards) of the head and neck of the bone upon the top end of the shaft of the femur. This rotation necessitates an inversion of the lower part of the extremity, if the cartilage of the head of the bone is to lie in contact with the cartilage of the acetabular cavity; in other words, when the head of the bone rests on the acetabulum the toes are inverted. This inversion of the foot predisposes to reluxation.

This rotation of the femur is an important factor in prognosis therefore. It is universal on one side in every bi-lateral case, the result being that in bi-lateral congenital dislocations it is impossible to get an anatomically perfect result on both sides by Lorenz's method.

XLI.—*Case of Dilatation of Large Intestine, Sigmoid, and Rectum, beginning at the Hepatic Flexure, and ending at the Pelvic Floor.* By H. Morley Fletcher, M.D. and Mr. H. Betham Robinson. *Exhibited April* 28, 1905.

Boy, æt. 12, admitted to the East London Hospital for Children, Shadwell, on November 15, 1904. Fairly healthy, but always had had a large belly and been rather constipated. A fortnight before admission there was a great increase in size, constipation more marked, and some pain on defæcation; no vomiting or passage of blood.

Examination showed a tense abdomen, with resonant percussion note all over; visible peristalsis. On palpation irregular masses felt in the region of the transverse colon. Pulse and temperature normal. Weight 3 st. 4 lb.

As the obstinate constipation was not relieved by aperients and enemata gave poor results, abdominal section was performed on November 29.

The pathological condition above stated was found, the upper part of the rectum having a circumference of 19¾ inches.

By manipulation bowels were emptied, many pounds of soft dark fæces being forced out *per anus*. Abdomen closed. Subsequent result was most satisfactory.

XLII.—*Case of Scleroderma of the Hands, Wrists, and Face, associated with Vasomotor Phenomena.* By John Fawcett, M.D. *Exhibited April* 28, 1905.

Albert C., æt. 21, a soldier, was admitted into Guy's Hospital in January, 1905, for "cold" hands, with inability to close them completely. The patient had first noticed that his hands became cold on exposure in May, 1904, and in the following September that his fingers were becoming "thick and stiff," with increasing difficulty in closing them. This condition gradually progressed until his admission to hospital. The patient had had no previous illness of importance nor any venereal disease. Although he had been in

South Africa during the war he had not undergone any severe exposure.

The hands have a permanently bluish-white tint and look cold. On exposure they become blue and remain so until they are warmed again. No pain nor any paræsthesiæ are felt in the hands, with the exception that slight tingling is experienced in them as they become warm after being cold. The skin of the hands, and that covering the lower third of the forearms is smooth, tightly stretched over the surrounding parts, and is of leathery consistence. This condition of the skin is most marked over the dorsum of the hands and fingers, the latter having a tapered outline from the stretching of the skin over them. The nails are more convex than usual from side to side. The fingers are kept partially flexed, and can only be bent about half-way into the palm. There is no ulceration of them, nor has there been any so far.

The hands become very moist when warm. The pulse at the wrists is small and very readily compressible. The colour of the face is similar to that of the hands. When patient is cold the ears, nose, and lips, which are usually of a dead white tint, become bluish. The skin over the malar bones and that over the nose, ears, and forehead has a smooth and somewhat shiny appearance, and is also somewhat more tightly stretched over the subjacent parts than usual, though not to so marked a degree as that over the hands and forearms.

The facial condition has developed to some extent since admission, so that the general expression of the patient has become more cadaveric, the increasing tightness of the skin tending to accentuate the hollows of the cheeks and to render the outlines of the bones more prominent. The feet are not affected.

Since the commencement of the disease there has been a considerable failure in the nutrition of the patient. He states that in November, 1904, his weight was 9 st. 6 lb. On admission in January, 1905, his weight was 8 st. 6 lb., and in May 7 st. 11 lb. No tenderness of nerves is to be detected anywhere, nor any loss of sensation. The only muscles in which the electrical reactions were doubtful were the first and second interossei of each hand, in which, although the muscles reacted to the faradic current, yet to the galvanic current there was a change in the polar reactions of the muscles in the left hand, A.C.C. being equal to K.C.C. In all the other muscles of the hands, forearms, and arms the electrical reactions were normal.

No evidence of organic disease was detected elsewhere in the body.

Remarks.—Although possibly this case may belong to the same category as Raynaud's disease, yet there have been no recurrent paroxysms and no pain. The condition has been a gradually progressive one associated with a general failure in nutrition. Apart from the somewhat doubtful electrical reactions in the interosseous muscles of the left hand, there is no evidence of any lesion of the central nervous system. The condition suggests rather an obliterative arteritis leading to atrophy of all the structures of the extremities and so far affecting chiefly the skin and subcutaneous tissue. Thyroid gland, potassium iodide, and nitro-glycerine have been administered, but without any material benefit. Massage and galvanism have been used regularly throughout.

XLIII.—*Case of Kaposi's Disease.* By JOHN FAWCETT, M.D. *Exhibited April* 28, 1905.

LEILA M. W., æt. 16, was admitted into Guy's Hospital in March, 1905, under Sir Cooper Perry, and later came under my care. Freckles were first noticed at 2½ years of age. From 3½ to 10 years the patient was the subject of corneal ulcers. Up to the age of 14 no lesion except those mentioned above had been noticed.

During the past two years many small sores have appeared on the face at intervals, which on healing have been replaced by thin depressed scars. About two months previous to admission in January, 1905, the tumour at the left side of the nose appeared, and has gradually increased in size.

There is no history of any similar affection in any of the patient's family.

On admission.—A protuberant, firm, fleshy mass was seen to involve the left ala of the nose, the adjoining part of the cheek and upper lip, and to almost completely block the left nostril. The surface, which was granular and ulcerated, emitted a foul-smelling discharge. The upper margin of the tumour was bounded by a hard everted edge of thickened epithelial and subepithelial tissues. The mass measured 2 inches in its longest diameter by half that in width.

Numerous freckles were seen on the face extending down

over the neck to within a short distance of the clavicles in front and as far as the spine of the scapula behind. Freckles also present over the usual situations on the arms, and to a lesser degree on the front of the legs. On the face are many thin, irregularly outlined, depressed scars, and some small round warts of a reddish tint. Telangiectases are also present. Some white atrophic patches are visible on the neck. There are corneal opacities in each eye. Some small, white, flat-topped warts present on the left arm and hand.

Course.—Sir C. Perry suggested the application of X-rays to the granulomatous mass occupying the left nostril, and this treatment has been carried out daily from March 28 to the present time. The result has been most striking. The mass has slowly diminished, so that now there is scarcely a trace of the projecting tumour to be seen. It consists at the present time of a shallow, flat, irregular ulcer measuring ¾ inch in its longest diameter and ¼ inch in width. The upper edge is a little thickened and everted, but is not more than one third as thick as when the patient was admitted. The nostril is quite clear.

It has not been possible to obtain a portion of the tumour for microscopical examination.

(This patient was exhibited at a meeting of the Dermatological Society on April 12, 1905.)

Additional notes.—(The following notes have been kindly given me by Mr. G. Sichel, under whose direction the X-ray treatment was carried out.)

Treatment with the X rays was commenced on March 28, 1905, and administered, at first daily, and later every other day. Applications were made for ten minutes at a time with 1–1½ tenths of a milliampère in the secondary circuit. Slight erythema and itching of the hands developed on April 21, so treatment was discontinued until April 25. From May 6 to May 26 the treatment was interrupted owing to an attack of measles. Previous to the onset of the measles seventeen applications had been made to the nose and eight to the hands. From May 26 to June 16 the X-ray applications were made daily, on one day to the nose, the next day to the hands, and the next to some of the warts on the face.

The tumour on the left nostril had entirely disappeared when patient left the hospital on June 16; vascular pterygium were less marked and some of the warts had disappeared.

The tubes used were rather soft ones, the alternative spark gap varying from 3½–1 inch.

XLIV.—*Tuberculosis of the Parotid Salivary Gland.*

By Douglas Drew, B.S. *Exhibited April* 28, 1905.

T. H., æt. 9, male. Father died of consumption.

History.—The swelling of the parotid began simultaneously with discharge from the left ear, and had lasted seven months. A "polypus" was removed from the ear three months later.

P.S., March 22, 1905.—The swelling has the outline of the parotid gland, and it is hard and slightly tender. There is no evidence of enlargement of the lymphatic gland over the parotid or of the glands of the neck. Towards the anterior part there is a small fluctuating area to which the skin is slightly adherent. The orifice of Stenson's duct is normal.

March 28, 1905. *Operation.*—An oblique incision was made below the lobule of the ear—over the swelling—and the superficial tissues were raised off. The fluctuating area was a small abscess, after scraping it out; two fine tracks led from it into the substance of the gland; these appeared to be ducts of the gland. The lobules of the gland were hard and shotty and distinctly inflamed. A small portion was removed for microscopic purposes, and the wound was closed with drainage.

At the end of a fortnight the wound was completely healed and the swelling was somewhat smaller.

Microscopic report.—This is a most marked specimen of tuberculosis of the parotid gland. The section shows that the glandular tissue is everywhere being destroyed by tuberculous granulation tissue containing many giant-cell systems. There are several areas of commencing caseation throughout the section.

Remarks.—Tuberculosis of the parotid salivary gland must be extremely rare—it is the first example that has come under my notice. It is hardly necessary to add that the condition was not diagnosed. The diagnosis made was inflammatory swelling of the parotid gland, with the formation of an abscess; it was thought that the condition was possibly secondary to the otitis media purulenta which existed. The case is also of interest from the point of view of treatment. Should the gland be extirpated? As the swelling is considerably smaller since the operation, I have decided to wait and keep the case under observation.

XLV.—*Tumour of the Tongue in a Girl.* By JOHN R. LUNN. *Exhibited April* 28, 1905.

A GIRL, æt. 12, complains of a lump on the right side of the tongue, which she has had for six months; it causes no pain or inconvenience.

On protruding the tongue, one notices on the dorsum of the right half, situated half-way along the surface, a swelling over which the papillæ are markedly hypertrophied. The swelling extends backwards, shelving off and then rising into a similar one further back. On palpation the swellings are found to consist of thickened tissue, but there are no definite nodules. All the right half of the tongue is thickened. The right cervical glands are distinctly felt enlarged. The teeth are not typically syphilitic, the bridge of the nose is depressed, there are suspicious scars at the left angle of the mouth. She has old ulcers of the cornea (interstitial keratitis). The mother is alive and healthy; other five children are quite well; no history of miscarriages.

XLVI.—*A Case of Traumatic Stricture of the Œsophagus relieved by Electricity.* By T. D. SAVILL, M.D. *Exhibited April* 28, 1905.

MRS. M. C., now æt. 49, swallowed some Condy's fluid by mistake in the year 1878, and this was followed by extreme dysphagia and stricture. She attended at the London Hospital at this time, and subsequently at the Throat Hospital (Golden Square), where after ten weeks as an in-patient a bougie was at length successfully passed. The stricture, however, again contracted, so that two years later a small bougie could only be passed with difficulty. Subsequently she learned to pass a bougie (No. 15 Downes) herself, and after passing the instrument before each meal she could swallow liquids and finely-divided solids, though often with considerable trouble.

The patient first came under Dr. Savill's care in June, 1903, with a history of gradually increasing difficulty, so that for the last few months no bougie could be passed and no

solids had been taken. For nine weeks she had only been able to take about one pint of beef-tea and one pint of milk in twenty-four hours slowly when very hot and whilst she was lying down; and during that time she had lost 14 lb. in weight. She had seen several surgeons at different times, and gastrostomy had been mentioned. Electricity was started on June 15, 1903, and improvement began after the third sitting. In six weeks, after twelve sittings, she began swallowing minced fish, meat, and fowl, could pass her bougie again, and had gained 7 lb. in weight.

The improvement was maintained without further treatment for eighteen months, when the same difficulty returned, and on April 4, 1905, the same treatment was instituted, with the same prompt effect, so that now, after eleven sittings, she can again take finely-divided solids without passing a bougie.

The electrical treatment consisted of the constant current (10, increased to 15, milliampères), applied for ten minutes, immediately followed by faradism applied for four to six minutes, the positive pole being applied over the nucha and the negative by means of an intra-gastric electrode passed down to the stricture, which was 12 inches from the teeth. After the first few sittings both currents (de Watteville current) were applied at the same time. The sittings took place about twice a week for six weeks on the first occasion, and about three times a week for four weeks on the second.

Remarks by Dr. Savill.—The patient appealed to me in her distress to see if electricity would relieve her and obviate an operation; the suggestion was hers, not mine. On the assumption, based on the history, that the difficulty was probably due partly to muscular spasm at the seat of the stricture and partly to atonic dilatation of the gullet above the stricture, I applied the galvanic current to relieve the former and faradism to relieve the latter. The possibility of an electrolytic action of the galvanic current on the fibrous tissue of the stricture might be suggested by some observers. But whatever the explanation, there can be no doubt about the efficacy of the treatment. On both occasions the same difficulty was relieved by the same means, and no other remedies or measures of any kind were employed either by me or by the patient; all her other conditions were the same all the while, on both occasions.

XLVII.—*Case of Ununited Fracture of the Tibia and Fibula, treated by Hoefftcke's Walking Apparatus.* By CHARTERS J. SYMONDS, M.S. *Exhibited April 28, 1905.*

J. McQ—, æt. 45, was admitted into Guy's Hospital under my care in August, 1904, for an ununited fracture, with malposition of the lower third of the tibia and fibula and some fixation of the knee. The injury was treated outside and there was said to have been a fracture into the knee-joint. No evidence of this latter injury could, however, be discovered either by manipulation or by the X rays. The fracture was exposed, the deformity corrected, and the ends of the bones brought into accurate apposition. The adaptation was so good that a wire was considered unnecessary. Primary union resulted. As no union followed between the bones a second operation was performed. The ends were found lying near together, both medullary canals closed by new material, and there was no evidence of periosteal formation. The ends were refreshed, the medullary canals freely opened, a wire passed through and the surface brought into close contact. Again the wound healed by first intention, but after six weeks there was still no sign of bony union. At this time the man again pressed for amputation, as his leave of absence was approaching its termination. It appeared to me that if he could be made to walk there was still a prospect of union taking place.

On February 6, 1905, I asked Mr. Hoefftcke to fit his modified walking apparatus. With this the man was able to return to his duties, which he has carried on ever since. At first he took no pressure at all upon his foot. Gradually the amount permitted has been increased. At the present time, April 28, it will be seen that although there is still movement in an anterior-posterior direction, the union is steadily improving. It has been particularly marked during the last fortnight. The knee has also improved in movement, the fibula has distinctly thickened, and there is sign of periosteal growth in the seat of the fracture.

The outlook for the man seems to be a good one, and there is every probability of his being able to dispense with the apparatus in the course of six months.

XLVIII.—*Multiple circumscribed Fibrous Masses of the Corpora Cavernosa.* By CHARLES GIBBS. *Exhibited April* 28, 1905.

F. C., æt. 48, late A.B. seaman. In June, 1904, during sexual connection patient hurt his penis and heard a distinct "click." Since then there has been impotence owing to upward chordee and acute pain at the site of the accident, which is in the dorsum of the corpora cavernosa in the mesial line about two inches behind the corona glandis.

In October, 1904, there could be felt a hard solid mass, square, with rounded angles, flattened from above downwards, about one inch in diameter. It felt like a mass of cartilage with very distinct edges. Running back from it was a hard, dense cord about the size of a leather shoe-lace.

In January, 1905, this mass was less clearly defined, much less hard. In the right corpus cavernosum there was a solid india-rubbery mass, reaching from 1¼ inches behind the glans to the perineum; the whole dorsum of the corpora cavernosa and string-like cord were more solid, tough, and india-rubber-like than they were; no glands could be felt.

In March, button on dorsum is less defined, smaller, and softer; the corpora cavernosa are less solid and tough except in the scrotal area. Upward chordee does not occur, but on rapid erection no distension by blood occurs in the pendulous penis, which remains flaccid; there is great swelling and discomfort in the proximal part of the penis; if erection be slow in its onset there is some partial erection of pendulous penis.

April 19.—There is a large mass, the size of a filbert, in the scrotal portion of each corpus cavernosum; they are hard, dense, rounded masses of fibrous tissue; the dorsal "button" is less defined. Erections as before, painful and partial.

Past history.—"Cut for stone when a baby"; no apparent scar now; has never ejaculated semen during intercourse; it passes into bladder, being subsequently micturated; has produced one child by this process. No syphilis, no gout, no diabetes; small septic sore thirty years ago.

Treatment.—anti-syphilitics, alkalies, and other gout remedies. Result, *nil.*

XLIX.—*A Case of Traumatic Cephalhydrocele.* By T. Crisp English, B.S. *Exhibited April* 28, 1905.

F. L., boy, æt. 8½.

History.—When six months old fell distance of 12 feet downstairs on to stone floor below; unconscious for three hours; no bleeding from the ears or nose, no convulsions. About five hours after the accident a swelling suddenly and rapidly formed on the left side of the head; this swelling has persisted ever since, though now somewhat smaller in proportion to the size of the head than formerly. The boy has always been bright and intelligent, and has never had fits or any other symptoms referable to the head injury until the last four months, during which time there has been occasionally some neuralgic pain about the swelling: bodily health excellent. No treatment at any time.

Present condition.—Intelligent, healthy-looking, and well-developed boy. Marked asymmetry of the head owing to abnormal prominence of the left parietal region: an elliptical opening is readily felt in the left parietal bone running from a point 2½ inches vertically above the auditory meatus towards the lambda, and measuring 4 inches in length and 2 inches at its widest part; the bone surrounding this gap forms an elevated and irregular ridge. The gap is occupied by a soft swelling which fluctuates and visibly pulsates; on firm pressure the swelling is partially reducible, and it is felt that the opening in the dura mater is much smaller than that through the superficial part of the bone. The swelling does not project sufficiently above the bony margin to allow examination for translucency. Skiagrams show accurately the extent of the aperture in the bone, but show no evidence of ossification in the floor referred to above.

Owing to the absence of any serious symptoms, it had been decided that no operative measures should be attempted.

L.—*A Case of Descent of the Left Cord, the Testis being retained in the Abdomen.* By W. G. SPENCER, M.S. *Exhibited April* 28, 1905.

THE patient, æt. 21, has never felt, either in the left scrotum or groin, a testis like the right one. In the upper half of the left scrotum a soft mass hangs down, which is drawn up a little when he coughs. It appears to be a patent funicular process of the tunica vaginalis, which becomes distended

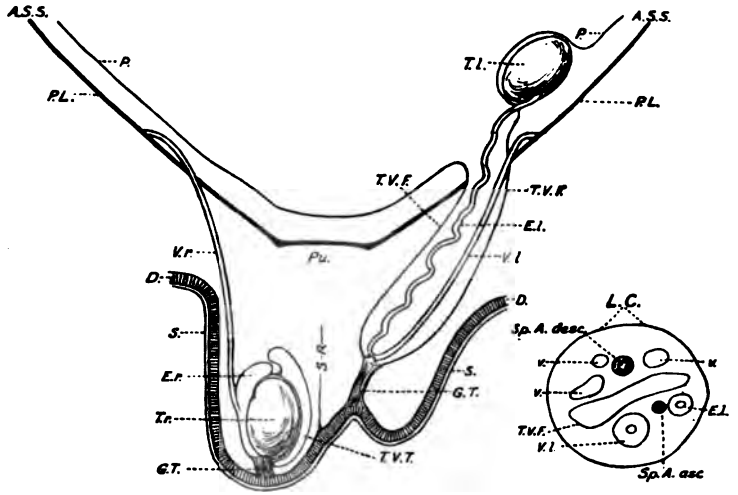

FIG. 4.—DIAGRAM ILLUSTRATING THE DESCENT OF THE LEFT CORD, THE TESTIS BEING RETAINED IN THE ABDOMEN.

A.S.S. Anterior Superior spine.—P.L. Poupart's ligament.—PU. Pubes.—S. Scrotum.—D. Tunica dartos.—S.R. Scrotal median raphé.—P. Parietal peritoneum.—T.V.F. Funicular process of the tunica vaginalis.—T.V.T. Tunica vaginalis testis.—T *l.* T *r.* Left and right testis.—E *l* E *r.* Left and right epididymis, the left unravelled.—V *l* V *r.* Left and right vas deferens.—G. T. Gubernaculum testis, on the left attached to the loop of the cord and to the fundus of the funicular process of the tunica vaginalis, so that when the latter was drawn upon the scrotum was infolded.—L.C. Diagrammatic section of left cord.—SP.A. *desc.* SP.A. *asc.* Spermatic artery descending and then re-ascending.—V.V. Pampiniform plexus of veins.

with fluid when the patient gets about, but the descent of a hernia has not been detected.

Addendum.—On incision a patent funicular process of the

tunica vaginalis was exposed, connected with the abdominal cavity by a long neck, passing through a well-formed spermatic canal without any sign of hernia. In the wall of the sac was found the vas deferens, of normal size, which, at its lowest end, was sharply reflected, and was then continued upwards as an unravelled epididymis forming a tortuous tube about half the thickness of the vas. This unravelled epididymis was traced up in the neck of the sac, to end in the testis lying just within the abdominal ring. The testis was rather more than half the size of the right, softer, and without a normal epididymis. At the angle made by the loop was attached a band of white fibrous tissue, about two inches in length, representing the gubernaculum testis. The other end of the band was continuous with the tunica dartos, so that as the sac was drawn upwards the scrotum, at the point of attachment, followed, and was drawn inwards. The drawing illustrates the condition. I operated a few years ago on a precisely similar case. In both there were two uncertain points, whether a painful lump of which the patient complained could be felt in the left iliac fossa just above Poupart's ligament, and whether a double vas could be felt in the scrotum. The patient was sent into hospital as a case of congenital hernia with a left atrophied testis in the scrotum, and many of those who examined him seemed to agree with that diagnosis.

LI.—*Congenital Absence of Lateral Abdominal Muscles, with Enlargement of Bladder and Ureters.* By C. Bolton, M.D. *Exhibited April* 28, 1905.

THE patient is a male infant, æt. 3 weeks, born at term, and the youngest of eleven. No other deformities have appeared in the family and, except in the abdomen, the child presents nothing abnormal. The recti muscles are present, but the lateral abdominal muscles appear to be absent, and the abdominal viscera can be palpated with ease. Distended coils bulge out the abdomen at the sides, and can be seen in outline, being placed immediately under the skin. They appear to be the distended ureters, intestine resonance not

being obtained over them. The bladder can be felt as a pyriform tumour almost reaching to the umbilicus and can be felt to contract; after being emptied a fulness can still be felt in this position. The prepuce was in the condition of phimosis, so circumcision has been performed. The testes cannot be felt in the scrotum. The umbilicus is normal; it bled a little on one occasion. The diaphragm and remaining muscles are normal.

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PRINTED BY ADLARD AND SON, LONDON AND DORKING.

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